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USE OF UREA IN TREATMENT OF CHRONIC OTITIS MEDIA

PRELIMINARY REPORT

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The essential features of almost every method of treating chronically infected middle ears are the observance of scrupulous cleanliness and the addition of some bactericidal agent.

Cleanliness is necessary to remove the necrotic débris, which retains moisture, blocks drainage, produces adhesions by its irritative action on delicate tissues and forms a fertile soil for the many types of bacteria and fungi which often complicate chronic otitis media. The use of an antiseptic is of secondary importance.

In attaining the two objectives one finds almost insurmountable difficulties. Adequate mechanical cleansing of all the recesses of the middle ear is often difficult if not impossible. The procedure is frequently painful and is always attended with a certain amount of trauma, which sometimes causes it to defeat its own ends. It must be repeated so often by a physician that many patients because of living at a distance or for economic reasons are unable to continue the treatment.

Many if not most of the antiseptics used, to be of any bactericidal value on mucous membrane, must be of such strength as to be severely irritating or damaging, which often in itself augments the discharge and hinders healing.

One or more of these factors occur so often as to make failure not infrequent, with discouragement to both the patient and the physician, leading to the continuation of a dangerous condition on the one hand or to radical surgical operation on the other.

In a recent article by Holder and MacKay [1] on the use of urea in the treatment of infected wounds, I was struck not only by the remarkable results they obtained when other measures had failed but by the fact that this was a modality which would be ideally suited to the treatment of many types of chronically infected ears. They said: "The mechanism of urea therapy is probably in part by virtue of the bacteri-

1. Holder, H. G., and MacKay, E. M.: The Use of Urea in the Treatment of Infected Wounds, J. A. M. A. **108**: 1167 (April 3) 1937.

cidal effect of strong urea solutions but chiefly through the solvent action on proteins, which leads to the removal of débris, incrustations and dead tissue, which mechanically and by harboring bacteria are the chief deterrents to normal healing." Here in a sentence is the solution to the problem of many chronic aural conditions.

In addition, they found, as have many other experimenters, that urea almost immediately relieved most of the odor of foul chronic wounds. Miller [2] used it to relieve the odor of sloughing cancer. Symmers and Kirk [3] found both clinical and experimental evidence of its action on putrefactive tissue. Péju and Rajat,[4] in a study of its bactericidal value, found it effective in weak solutions against many organisms, especially Bacillus diarrheae, Bacillus psittacosis, Bacillus pyocyaneus, Bacillus dysenteriae, Bacillus enteritidis and others producing a foul odor. Much of this was confirmed by Foulger and Foshay,[5] who found it to be of value against many strains of streptococci.

There are many reports attesting to its mildness and to the fact that it is almost totally nonirritating to living tissue. Symmers and Kirk [3] used it in dressings over Thiersch grafts, and this was repeated several times by Holder and MacKay.[1] I have never found that it harms the delicate epithelium of a radical mastoidectomy cavity and have used it on exposed dura without signs of irritation or toxicity. Occasionally, on acutely inflamed or raw tissue there is some pain, but this is seldom comparable with that produced by alcoholic solutions.

Foulger and Foshay [5] used urea as an antiseptic in a limited number of acutely infected middle ears of children and found that the odor was eradicated and the course much briefer than was to be expected.

With this in mind, I began studying its effect on a number of patients with chronic disease of the ear, and was more than pleased with the results.

The first patient had a radical mastoidectomy cavity, which had been unpacked a few days previously. Despite daily cleaning, it had a foul irritating discharge, which crusted on exposure to air. The cavity was dusted with urea crystals once, which eradicated all odor within twelve hours. After this, gross amounts of the crystals were applied twice daily by the patient, which resulted in a cessation of all discharge in a few days. The granulation, which I had often found to be stimulated by other antiseptics, seemed to be retarded and of a more normal pink, with a resulting increase in the rate of epithelization. The cavity was completely healed in less than five weeks.

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2. Miller, W. M.: Urea Crystals in Cancer, J. A. M. A. **100**: 1684 (May 27) 1933.

3. Symmers, W. St. C., and Kirk, T. S.: Urea as a Bactericide, and Its Application in the Treatment of Wounds, Lancet **2**:1237, 1915.

4. Péju, G., and Rajat, H.: Note sur le polymorphisme des bactéries sans l'urée, Compt. rend. Soc. de biol. **61**: 477, 1906.

5. Foulger, J. H., and Foshay, Lee: The Antiseptic and Bactericidal Action of Urea, J. Lab. & Clin. Med. **20**: 1113 (Aug.) 1935.

I have treated all my patients who underwent radical mastoidectomy in a similar manner, with equally pleasing results. It has eliminated almost altogether the necessity of daily packing, punching and scraping, which often goes with the postoperative treatment of a radical mastoidectomy cavity. In one cavity showing a large area of dura I began cautiously with dilute solutions of urea, gradually working up to a saturated solution. There was no evidence of irritation at any time, but I should advise caution in similar cases until the limitations of the drug have been determined.

I have tried the treatment on several patients who came in for cleaning of radical mastoidectomy cavities and found that a week's use of a solution of urea cleaned out almost all débris, leaving the skin pink and healthy.

In chronically infected middle ears in which the saturated solution could be brought in contact with the diseased area, I have found similar satisfactory results with few exceptions.

An elderly woman with chronic otorrhea of twenty years' duration without symptoms showed sclerosis of the mastoid in roentgenograms. There was a scant foul discharge from a large perforation, with necrosis of much of the malleus. Cholesteatoma could be removed. She definitely refused any operative treatment, which was not urged because of her age and lack of symptoms. I felt that the aqueous solution of urea might cause swelling of the cholesteatoma; so a small amount of the crystals was dusted with a powder blower into the ear twice daily. This is safe, as urea tends to absorb moisture from the surrounding tissue. Within two days the odor was eradicated, and within ten days the ear was dry and clean. A small granulation had subsided and become epitheliated; the necrotic area of the ossicles was smooth and clean, and though probing showed enlargement of the epitympanic region no cholesteatoma could be found. She is being carefully followed, but the ear has remained dry for over a month at the time of writing. I am, of course, not advocating this as a treatment for cholesteatoma when surgical intervention is indicated.

Eleven other persons with chronic otitis media with large perforations were treated by dropping a saturated solution of urea in the ear twice daily. The odor was rapidly eradicated, and all the ears are dry and clean at the time of writing. One had been treated several times by ionization, without success, and many had had several different courses of treatment, with only temporary relief at best.

One patient showing a large granulation failed to respond to treatment until this had been removed, after which the discharge subsided, and granulations failed to recur after the use of the aqueous solution of urea. Failure in this case was due to the fact that drainage was being blocked and the medication could not successfully reach the diseased area. Similarly, in cases showing a small perforation, results have not been good and I felt that if failure continued I should be justified in enlarging the perforation.

Contrary to the experience of Foulger and Foshay,[5] I have not had entirely satisfactory results with urea in a limited trial for acute otitis, nor is the rationale of the use of the treatment as clear as with chronic processes. Similarly, I have had no success in averting the conditions which progress to acute mastoiditis, for it seems hardly possible that the medication can be brought in contact with any considerable portion of the diseased area without the use of dangerous methods. However, I have found no contraindications to its use, and in the hands of those who have sufficient patients to be of statistical value it may prove its worth. I should caution, as Foulger and Foshay did, that caking of the urea may block a small perforation or the canal, but this is easily removed by a few drops of sterile water, which readily dissolves the crystals.

TECHNIC

My experiments would hardly justify, nor do I want to suggest, any inflexible technic in so new a procedure. To avoid complicating the results I have used urea alone satisfactorily, although I see no reason why it should not be combined with any form of therapy which the individual physician might favor or the variation of the condition warrant, with even greater success. Indeed, the work of Holder and MacKay on various combinations of urea with other forms of therapy is of interest and will undoubtedly be of value in selected cases of aural disease.

I have applied the solution with a dropper twice daily, beginning with small amounts and increasing rapidly if there is no pain. The crystals may be applied directly or with a powder blower, care being taken to remedy any caking either in the canal or in the powder blower. A small amount of water or saline solution will usually correct this.

In a few cases there will be some pain at first. I have attempted to relieve it by adding various local anesthetics, but this is not an entirely satisfactory procedure. Perhaps the use of urea phenolate, which is being developed, will be the answer. However, the pain has seldom been prohibitive and usually subsides in a day or so.

Foulger[6] suggested that urea be used cautiously in cases in which there is danger of hemorrhage, for it may dissolve a clot. So far, I have had no difficulty of this kind, but it seems a reasonable warning.

Experimentally, the solution will slowly dissolve cholesteatoma, but, like any aqueous solution, it tends to make the dried tissue swell a little and for this reason must be avoided or used carefully. The crystals, while less satisfactory, are entirely safe.

In closing, I urge the physician in using urea not to overlook a neighboring pathologic process or factors in the general health which

6. Foulger, John: Personal communication to the author.

often play a dominating rôle in prolonging infection anywhere, for I am convinced that urea therapy is not a cure-all or a new magic healing power but that the results are simply due to a more adequate removal of the gross and microscopic débris in the recesses of the middle ear, giving nature a fair chance, often with surprisingly successful results.

SUMMARY

I am quoting in part much of what Holder and MacKay said.

1. Urea in aqueous saturated solutions or in crystalline form by its power of dissolving necrotic débris removes the chief deterrent to healing in many types of chronic aural infection.

2. It is almost totally nonirritating to living tissue.

3. It is mildly bactericidal to many forms of organisms, especially those producing putrefaction.

4. It almost immediately eradicates all odor without producing another odor.

5. It is inexpensive.

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MEASURING THE SENSATION OF LOUDNESS

A NEW APPROACH TO THE PHYSIOLOGY OF HEARING AND THE FUNCTIONAL AND DIFFERENTIAL DIAGNOSTIC TESTS

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It has been assumed that similar sounds should appear alike to all ears with equal acuity of hearing as determined by the standard tests, that the loudness of a sound always corresponds to the number of decibels above minimum audibility and that the sensation of loudness may be predicated with confidence on the usual functional tests for hearing. I shall demonstrate the fallacy of all these assumptions.

In the past all methods for measuring acuity of hearing have been based on the ability of the ear to detect the faintest audible intensity. In every case it has been taken for granted that if the hearing is deficient for faint sounds it is also in like proportion deficient for louder sounds. In other words, if the hearing is down, say 30 units, at any or all intensities, it will require 30 extra units at any or all intensities to make it sound as loud as to a normal ear.

It will be shown that for purely obstructive lesions this assumption is generally true but that for so-called nerve deafness (lesions of the neural apparatus of hearing) this assumption is not true. I observed this phenomena over twelve years ago and reported it before the American Otological Society,[1] but only lately has its significance been recognized.

The ear is accustomed to listen to fairly loud sounds. It usually ignores faint sounds; in fact, special effort is needed to sense very faint sounds, and even with concentrated attention they are heard intermittently. Threshold sounds appear and disappear from consciousness because many external and internal factors interfere with detection. Loud sounds, on the contrary, are compelling; they are sensed willy nilly and in spite of inattention.

Perfect hearing of speech is impossible at minimum intensities. The best hearing for articulate sounds is normally attained at an intensity of about 50 to 60 db. (decibels) above threshold. This is the usual

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1. Fowler, Edmund P.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. **8**:151 (Aug.) 1928.

intensity of ordinary speech at a distance of about 4 feet (1.2 meters). At greater distances, unless the speaker raises his voice (which he usually does automatically), words or phrases are frequently missed or misunderstood even when the hearing is perfect. When there is any deafness, distance increases the percentage of missed or misunderstood words or phrases, because the loudness of a sound diminishes directly as the square of the distance, and the loudness of the speech sounds is then far below the customary requirements. On the other hand, many persons who have a marked loss in hearing for distant sounds or for faint sounds hear near-by conversation well, often quite as well as those with normal hearing. If the speaker raises his voice, such persons often complain and say: "Please don't shout at me. I can hear you perfectly." Strange to say, they may hear perfectly if the words are spoken at the common intensity of 60 db. above the normal threshold.

One reason for this apparent paradox is that in these cases there is a greater sensation of loudness in the deafened ear than has been suspected. The sound appears so loud that to the deafened ear it may sound as loud as to the normal ear, i. e., in an ear in which the hearing is down some 30 db. the sensation when the intensity is raised 30 db. (to a total of 60 db.) may be equal to that in the opposite normal ear at the same intensity. With monaural losses of 60 db., increase of the loudness to 90 db. above normal threshold may bring about a disappearance of the difference in loudness in the two ears; i. e., in spite of marked deafness in one ear (60 db.) and in spite of no deafness in the opposite ear, there may be no difference in the sensations of loudness for sounds 90 db. above the normal thresholds.

This at first thought appears impossible, but it is a fact substantiated by hundreds of tests of my own and by confirmation in the acoustic research laboratories of the American Telephone and Telegraph Company by Fletcher and Steinberg, and by several other physicists, physiologists and persons experimenting with animals. Dr. Lorente de Nó [2] stated: "It is what should have been suspected from the way that all nerve mechanisms work." I cannot do better than to quote his words:

"Dr. Fowler has observed that in patients with unilateral nerve deafness the difference in sensitivity of the two ears diminishes when the intensity of the testing tone is increased, so that with high intensities the loudness becomes equal for both ears.

"This indicates that when the intensity of the tone is high, the deaf ear, in spite of defective nerve mechanism, is sending to the acoustic

2. The following paragraphs are based on material from verbal and written communications from Dr. Lorente de Nó to the author.

centers very nearly the same number of impulses as the normal (or better ear)."

The central nervous system receives from the peripheral end-organs rhythmic impulses in series by way of the nerve fibers which together constitute the cochlear division of the auditory nerve. Each fiber conducts an impulse of uniform duration and pattern. The frequency in the individual fibers as well as the number activated determines the intensity of sensation from the peripheral stimulation. Weak stimuli activate only a small number of cells of the peripheral end-organ and therefore only few nerve fibers are set in action to conduct the impulses, in addition to which the frequency of the impulses in any one fiber will be low. With an increase in the stimulus, both the number of acting fibers and the frequency of the impulses in the previously acting fibers increase, and therefore the central nervous system receives a larger number of impulses per unit of time.

"With identical loud intensities, the loudness becomes equal for both ears, because the deaf ear in spite of a defective neural mechanism is sending to the auditory centers the same or very nearly the same number of impulses as the normal or better ear. If the frequency of the impulses set up in a sensory nerve fiber were strictly proportional to the strength of the stimuli (intensity of sound) no easy explanation could be found for the Fowler phenomenon, but this is not the case, because the frequency is limited by the period of recovery after each single impulse."

This period during which the nerve is unable to conduct impulses is called the refractory period. It varies from fiber to fiber but is always present, so that for each fiber there is a limiting frequency of impulses which cannot be exceeded.

"The shortest refractory periods recently observed by Gasser and Grundfest, and by Graham and Lorente de Nó, are in the neighborhood of 0.0005 second, so that 200 impulses per second is the highest possible frequency of impulses that can be maintained by the fastest nerve fiber, and that for only very short periods of time."

In the cochlea each nerve fiber comes in contact with a number of cells of the sensory end-organ (the internal and external hair cells), and therefore, even if only a few of these cells are strongly stimulated, they will be able to saturate the nerve fiber which innervates them and the same number of impulses will proceed from the ear with a number of hair cells missing as from the normal ear. In other words, at high intensities of stimulation the limiting, or saturation, excitation will be obtained as well when a number of hair cells have disappeared as when all the hair cells are present unless there is widespread degeneration of the hair cells: a block destruction.

In the central nuclei in a somewhat similar way, the cochlear nerve fibers also divide into branches which form special endings (synapses)

on a number of cells, so that each nerve cell receives endings (connections) from several nerve fibers. Furthermore, there are association neurons in the central cochlear nuclei, and these take care of summation effects from nerve impulses arriving during a given interval. If a number of fibers of the cochlear nerve are missing, many of the neurons in the cochlear centers will be stimulated below threshold, by sounds of low intensity, whereas in the centers connected with the normal ear these same neurons will be responding in full to the stimulation. Hence there will be a difference in the degree of hearing at or near minimum audibility in the ears in question.

If, however, the intensity of the sound is increased, the neurons on the affected side (the deafened ear) will be stimulated just as strongly as those on the normal side, because both will be receiving the maximal, or "saturation," stimulation which takes place when all the synapses on any central limited zone of the body or the dendrites in that zone are simultaneously active (Lorente de Nó, 1935). It follows that the higher centers (cerebral cortex) will receive the same number of impulses from both the normal and the nerve-deafened ear and the same sensation of loudness in the normal as in the diseased ear.

"In short, the Fowler phenomenon has to be interpreted as a logical consequence of two well known facts, the overlapping of the peripheral and central end arborizations of the cochlear fibers and the existence of a maximal (or 'saturation') stimulation for both nerve fibers and nerve cells."

To utilize the phenomenon as an aid to diagnosis, one should throw into the normal ear (or better ear) loud sounds—10, 20, 30 decibels or more in excess of the intensity required for minimum audibility in the poorer ear—and then throw into the poorer hearing ear the same frequency at an intensity which appears to the patient to be of equal loudness (i. e., balance the loudness in the two ears).

If it requires a markedly smaller number of decibels to bring the deafened ear up to the same sensation of loudness experienced by the better ear than is required near the thresholds, it means that the difference in the hearing of the two ears at thresholds is not due to an obstructive lesion. It means that the difference in hearing is due to a neural lesion in the poorer ear.

On the other hand, when there is no recruitment of loudness in the poorer ear, i. e., no change in the differences in hearing at the thresholds required to balance the loud sounds binaurally, it means that there is an impedence (conduction) lesion in the poorer ear. At all intensities lesions interfering with conduction maintain the same differences in hearing between the two ears. A normal ear or an equal binaural lesion in the nerve will also show no differences with changes of intensity. It

is surprising how accurately this test may be accomplished even with little children.

If double audiometer receivers are not available, a simple technic is first to determine the difference in hearing of the two ears at thresholds, and then to compare binaurally a loud sound (say 80 or 90 db. above the normal threshold) by placing the receiver first over one ear and then over the other. If in unilateral deafness this sound appears about equal to the two ears, it means that the difference in the hearing at threshold was caused by nerve deafness.

I have devised a simple method of plotting the results of the loudness balance test on my regular audiogram charts. These show its simplicity and great reliability as a means for measuring the sensation of loudness and for differentiating nerve deafness from conduction deafness.

There are other possibilities for usefulness, such as overcoming the inaccuracies of observation due to tinnitus aurium, differentiating tinnitus of the middle ear from that of the internal ear, and aiding in prescription of hearing aids for persons with nerve deafness. These will be reported at some later date. I call attention to the fact that this is the only reliable test for nerve deafness which does not require confirmation by bone conduction or other tests.

The phenomenon I have described may be useful also in the differential diagnosis of lesions affecting other sense mechanisms, because all nerve mechanisms act fundamentally in the same way. The methods I have described for measuring relative loudness may therefore with appropriate stimuli be used to measure the relative intensity of sensations mediated by other organs affected by a pathologic process (sight, touch, pain, the static sense, etc., may be measured in comparison with the normal or more nearly normal).

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ABSTRACT OF DISCUSSION

DR. WALTER HUGHSON, Abington, Pa.: Dr. Fowler's study offers a great possibility of filling a want long felt by practicing otologists. Since his report before the American Otological Society in Detroit in 1936, I have been much interested in the phenomenon both from a clinical and from an experimental standpoint. An investigation is now being carried out in my laboratory to study the effect on the response to loudness of specific experimental lesions both of the conductive and of the neural mechanism of the ear. The experimental technic is considerably more involved than that required for clinical tests, but one fact of importance has developed, which may or may not be significant. When the normal ear is exposed to sounds of high intensity (80 to 90 db. above threshold for any great length of time), overload and fatigue develop with great regularity. Such intensities are used in routine tests. In my small group of clinical cases there has been no instance of such fatigue, but in any considerable series it would surely develop at some time. Should fatigue take place, the result of the measure-

ment of loudness might be misleading. I feel, therefore, that tests should be made as a routine to determine whether or not fatigue has occurred.

It is unfortunate that the loudness balance method is not applicable to impairment of less marked degree than that in the cases mentioned by Dr. Fowler. In my hands the measurements are of no particular value unless there is impairment for all frequencies of at least 20 db. An expected error on the part of the patient of plus or minus 5 db. in any test of the kind would make an accurate differential diagnosis extremely difficult. In early deafness prognosis is of the utmost importance but is necessarily dependent on diagnosis. With further refinements in the methods of testing it will undoubtedly be possible to make diagnosis and prognosis for early monaural or unequal deafness as well as for rather advanced deafness.

To illustrate the point of overload and fatigue, I shall show the loudness curves for an animal for frequencies of 500, 1000 and 2000 cycles. Both ears were entirely normal. One ear was used for reference. As the intensity was increased, the ears responded in exactly the same way up to the maximum intensity of stimulation of 100 db. above threshold. If this were taken as the final word in this examination, I should consider that the ears were responding equally as the intensities increased. However, if the charting is done in a different way, it is found that at 80 db. above threshold the ears begin to overload and then promptly become fatigued. So if high intensities of stimulation are used on a normal ear, great care must be taken to ascertain whether or not that ear has become fatigued.

In another experiment in which the conductive type of deafness was produced on the experimental ear, the curve was exactly that which Dr. Fowler gets in his conductive type of deafness, but I find that the normal ear is fatigued and the ear with experimental conductive deafness is not fatigued at all. As the curve turns over, overload and fatigue are indicated.

Dr. Douglas Macfarlan, Philadelphia: In regard to Dr. Fowler's paper, two thoughts come to me. 1. This is a statement of a fact, the value of which I predict will be great when otologists begin to use it in clinical testing. 2. Dr. Fowler offers an explanation for the fact and substantiates his opinion with the opinions of others recognizing his finding. This explanation falls in the same category as the explanations of the physiologic processes of hearing. Otologists are doing more clear thinking on the theories of hearing than was done by the past generation, and this is due to the assistance of actual experimental findings. There is, however, a great deal that remains conjectural.

The high point of Dr. Fowler's address lies in the valuable suggestion it gives of differentiating catarrhal deafness from nerve deafness. More clinical work has to be done to establish this item of differentiation as a certainty, and there will be cases of mixed deafness which will throw confusion into the findings. Undoubtedly, attention will appear as important as it does in all cases of profound deafness, no matter of what nature. It will be interesting to know whether inattention actually produces incapacity through disuse and whether or not the Fowler phenomena will appear in cases of advanced and long-standing deafness that is apparently purely catarrhal.

Dr. Horace Newhart, Minneapolis: I have sufficient faith in the resourcefulness of electro-acoustic engineers to hope that through their experiments improved types of hearing aids ultimately will be developed which will enable the perplexed otologist to prescribe or select more efficient electric hearing aids for that large group of persons who are severely handicapped by their inability to perceive the higher frequencies. These persons are unable to tolerate the great

amplification and inevitable distortion produced by electric hearing devices employing carbon microphones. The condition is the despair of the practicing otologist.

Until more efficient portable devices are developed, possibly along lines suggested by the facts brought out by Dr. Fowler's paper, otologists must recommend to persons with severe nerve deafness the use of semiportable vacuum tube hearing aids. These have been greatly improved recently as regards compactness, lightness, fidelity of reproduction and other desirable features.

In the past few weeks my attention has been called to a new semiportable device by the unbounded enthusiasm of several of my patients who have recently thoroughly tried it with far greater satisfaction than they have had with any other of the many hearing aids they had previously used. Its high grade microphone and crystal receiver give it great fidelity; it can be operated at will either by its self-contained batteries or from the electric light current; it has a powerful induction telephone attachment whereby telephone conversation is greatly amplified without distortion, and it is economical in the use of batteries. The use of devices of this type, as soon as they are made available on a commercial scale by one or more manufacturers, should be recommended by the otologist to persons who cannot be satisfactorily served by the portable hearing aids now obtainable. The early employment of smaller, improved vacuum tubes already to be had abroad will make possible a further reduction in the size and weight of these remarkably efficient instruments.

Dr. Frederick T. Hill, Waterville, Maine: Dr. Fowler called attention to binaural loudness balance in a paper read at the meeting of the American Otological Society last year, suggesting as a possible method for early detection of otosclerosis observation of the hobble effect on loud tones. The paper just read has clarified things a good deal by utilizing the physiologic explanation of overlapping of fibers and of saturation stimulation.

One must differentiate between loudness and intensity. One may think of loudness as subjective, or sensory, and of intensity as objective, or motor. Increasing the intensity may make equal or balance the loudness, as interpreted in the two ears.

This alternate binaural loudness balance is applicable in cases of asymmetrical loss of hearing. If the perception of loudness by the two ears in such a case can be balanced, one may assume that the difference is due to a lesion in the nerve. I say difference, for there may well be coincident conduction deafness. If the sensation of loudness in the two ears cannot be made to balance, the condition may be considered as purely conduction deafness. In other words, positive findings are limited to asymmetrical nerve deafness.

One of the most practical applications seems to me to be in its possible utilization for more accurately prescribing hearing aids. There is great need for improvement over the present methods.

In nerve deafness, sounds, if loud enough, may be heard well. If amplification is uniform, faint sounds may be properly amplified, but the loud sounds may become disagreeable, even painful. This may explain some disadvantages of the instruments now used. It suggests a problem in selectivity.

I should like to ask Dr. Fowler if in his opinion the phenomena of paracusis are explainable on the basis of overlapping association fibers.

Dr. Edmund Prince Fowler, New York: I shall first answer one of the questions and then show a few slides to demonstrate how simple it is to do these tests. It is not nearly as complicated as it appears. The ears are accustomed to balancing the loudness of sound binaurally. That is how one tells the direction

from which a sound comes. Even stupid or inattentive persons cannot help balancing intensities binaurally. The loudness of the sound fixes the attention.

As far as the overloading is concerned, there is not nearly the fatigue from this method of testing, because the sounds are only momentarily applied. If such sounds cause an overload, any sound can cause an overload. Of course, one does get fatigue from any sound if it is continued long enough, but this test causes about as little fatigue as one can imagine. Even though the ears differ from each other 5 or 10 db. from hour to hour or from day to day, if one has nerve deafness of 5 or 10 db. the otologist can discover it more surely by this than by the bone conduction test, because the result of the bone conduction test may vary 15 or 20 db. up or down.

As to the phenomenon of paracusis, the recruitment of loudness phenomenon does explain why the hearing is worse in a noisy place in nerve deafness and with normal hearing and the reverse in obstructive deafness.

A TREATMENT OF CHRONIC PURULENT OTITIS MEDIA

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By using common sense and ordinary judgment one must conclude that the percentage of cases of chronic purulent otitis media in the population is much less now than it was two or three decades ago. There is room for improvement. Otologists agree that the chronically suppurating ear should be kept clean and little has been added to this one principle in a decade or two or three. During this time radical mastoidectomy, zinc ionization, treatment with silver nitrate and other procedures were at different times hailed as the answer. Hett, Wells and Levick [1] reported 75 per cent of cures with zinc ionization. Perhaps nearly as great a percentage could be obtained by keeping these same ears clean with dry cotton followed by the insufflation of boric acid powder. Lederman [2] laid great stress on thorough cleansing of the ear before iodine powder is used. To my mind, cleansing is the most important item in any method of treatment one may choose. Rodger,[3] in 1933, recommended and used irrigation in all chronically suppurating ears and wrote as follows: "The patient should be told that we are going to make a determined effort to get the ear dry by conservative measures, but that if we do not succeed within a reasonable time-limit we must assume that there is some deeper mischief and that operation will be necessary." Few now irrigate, and to most otologists the radical mastoidectomy is not a treatment for uncomplicated chronic suppurative disease of the middle ear.

In 1922 I saw a patient who presented all the results of an excellent radical mastoidectomy.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 10, 1937.

1. Hett, Wells and Levick, cited by Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **12**:390 (Sept.) 1930.

2. Lederman, M. D.: Twenty Years Experience with Iodine Powder (Sulzberger) in the Conservative Treatment of Aural and Nasal Suppuration, Laryngoscope **47**:110, 1937.

3. Rodger, T. Ritchie: The Treatment of Chronic Suppurative Otitis, J. Laryng. & Otol. **48**:525, 1933.

He was presented by a friend, a physician of the United States Public Health Service. The story was that a few years before the two of them were on a hunting trip in the desert mountains of Arizona. On the fifth day in camp the patient reluctantly told his friend that he was in great pain and that a chronically discharging ear had become infested with maggots. Dr. F. immediately took him 60 miles (96.5 kilometers), to the nearest physician. The physician was not in, but Dr. F. found in the office a 5 per cent solution of cocaine. Thinking to relieve his friend's pain he dropped some of the cocaine solution into the ear. Immediately there was an exodus of maggots, and all of them died immediately. I have described the result.

This incident gave me the idea of using a digestant to relieve chronic suppurative otitis media. I have used various digestants, but none have been so satisfactory as the solution of the extract of Carica papaya, or papaw.

For the past few years I have used the powdered extract of Carica papaya in chronically suppurating ears with satisfactory results. A solution made the moment before is instilled into the ear while the patient is prone and is allowed to remain for a few seconds, and then the ear is wiped dry. The solution penetrates to the far crannies better than powder. This vegetable digestant cleans the surface of visible pathologic tissue and after a few treatments, in the ordinary case, leaves a dry ear. The solution may be injected into the eustachian tube, and the results in empyema of the eustachian tube have surprised me.

Case 1.—M. H., a 16 year old girl, gave a history of having had a discharging right ear since her earliest recollection. Her sister, who was a nurse, said that the discharge had been present all her life. Treatment was begun on May 7, 1935, with powdered extract of Carica papaya. The first month the powder was insufflated twice a week after all discharge was wiped away. After the second month the ear was entirely dry, and a few times iodine powder was used and the ear continued dry until she left in April 1937. In the meantime she had had several severe colds with no discharge from the ear resulting. In this case the perforation was large and central.

Case 2.—Mrs. G. W. S., 35 years of age, presented herself for treatment on March 9, 1937. There had been intermittent discharge from the right ear all her life. The perforation was in the posterior third of the drum, and in it were some cholesteatomatous-appearing masses. The patient was complaining of dizziness and loss of hearing in the right ear. Roentgen examination showed marked loss of bony tissue in the right mastoid, which I attributed to cholesteatoma. The solution of Carica papaya powder was used several times with the patient lying flat on the left side, and the cholesteatoma in the middle ear disappeared. On Sept. 16, 1936, a radical mastoidectomy was performed on the right side. A large cavity was found, with the exposure of the dura in two places and of the lateral sinus in an area about 10 by 16 mm. The cavity contained no cholesteatomatous masses but was lined with a thin membranous material, and whether the cholesteatoma was digested by the solution is problematical. Recovery from the operation was uneventful, and at the time of writing the hearing was improved from 87 per cent loss to 48 per cent loss.

SUMMARY

My cases have been fewer than sixty and therefore are worth little from a statistical standpoint.

The powder may be insufflated and frequently is, but obviously powder blown into a perforation in the drum does not get very far.

I regard the drug as harmless since, so far as I know, it is purely a digestive agent. This treatment, being purely a "digester," so to speak, may offer something in the treatment of cholesteatoma.

ABSTRACT OF DISCUSSION

DR. SAMUEL J. KOPETZKY, New York: The essayist offers another procedure for the treatment of chronic suppuration of the ears and reports satisfactory results thus far. In observation in the trial of any procedure, the intense attention that the patient gets at that particular time may often lead one astray in evaluating the results that are obtained. There remains the fact that there are persons with chronic suppuration from various causes.

If it is proved that the digestant advocated can dissolve granuloma, that it digests and does away with the general stroma, the fat and the desquamated epithelium causing the mass of a cholesteatoma and subsequently that this digestive process removes the dead and dying bone of caries, then otologists are indeed indebted to Dr. Harris for bringing it to their attention.

All these chronic conditions are not necessarily live and active infections. Many of them are purely secondary saprophytic reactions which occur in the middle ear as a result of antagonism between different types of epithelium. One type of them dies, and another forms a mass which becomes saprophytically infected and then discharges pus into the canal of the ear.

I think that one should receive this paper with an open mind. All physicians observe patients with these annoying conditions and should give this method a fair trial.

DR. BENJAMIN H. SHUSTER, Philadelphia: I have had some experience with the use of a powdered extract of Carica papaya. It was suggested to me for use for chronic infection about the nose and in the ear. I used it a number of times, but, my results not having been brilliant, I discontinued it. Dr. Harris brings up the question again; probably a better method of using the solution might be of greater value. It is always worth while to try out a new idea. When iodine powder first came out, I am sure many physicians found a greater number of patients with uncomplicated chronic purulent otitis media improved as a result of its use. The drawback of conservative treatments is this: One probably overlooks two things: The first is that there are conservative surgical measures which are of great help. For instance, the operation suggested by Dr. Babbitt, a very simple procedure, cures a number of conditions rapidly merely by cleaning out granulation tissues in the attic. Secondly, in the enthusiasm and desire of the otologist to prevent the inconvenience to the patient of performing an operation, he may continue conservative treatment to the point of its becoming dangerous. No matter how much one digests cholesteatoma, I cannot conceive that in any case of chronic otitis media with chronic mastoiditis which manifests recurrent acute exacerbation, treatment by conservative measures would be worth while. The same is true in cases in which there are symptoms indicating necrosis of the bone, such as facial paralysis or persistent or frequently recur-

ring headache as a result of chronic infection. Conservative treatment may cause one to overlook a more serious situation.

I recall one case in which conservative measures, either surgical or medical, would have been of no avail. I treated an old man with a subacute discharge from the ear, and occasional pain in the head. I tried to treat him conservatively, hoping that the condition would clear up. Suddenly a severe headache developed, and I decided that something had to be done about the mastoiditis. I cleaned out the mastoid completely without finding sufficient pathologic involvement to account for such a severe headache with signs of meningeal involvement. I decided to expose the dura, and to my surprise, after chiseling through about ½ inch (1.25 cm.) of dense, ivory-like bone, I found a perforation through the tegmen into the middle fossa and free pus in that location. There are certain cases in which one must not let conservative measures create overenthusiasm causing dangers to be overlooked.

A SIMPLE TECHNIC FOR TAKING MOTION PICTURES OF THE LARYNX IN ACTION

JOEL J. PRESSMAN, M.D.
AND
ARTHUR HINMAN
LOS ANGELES

For the past three years we have been employing a satisfactory simple technic for photographing the larynx in action. The results of our earliest efforts were first shown by Dr. George M. Coates at the meeting of the American Academy of Ophthalmology and Otolaryngology in 1935.

The method to be described has three important features to recommend it. First, it requires no expensive or unusual apparatus; second, photographs are obtained with the area under local anesthesia so that laryngeal movements can be recorded, and third, the photographs are taken during routine examination or treatment, with a loss of time not exceeding one or two minutes.

Anesthesia is accomplished by the use of a 10 per cent solution of cocaine hydrochloride applied locally to the larynx as for any direct laryngoscopy or bronchoscopy. The patient reclines on the operating table; the head is held by an assistant, and the direct laryngoscope is inserted according to the method for direct laryngoscopy described by Dr. Chevalier Jackson. The details of the proper technic for exposure of the larynx by the use of the direct laryngoscope can be studied in any of the standard works by this author.

The source of light is the ordinary bulb on the Jackson-Pilling direct laryngoscope stepped up to the maximum intensity available on the Pilling bronchoscopic battery. These light bulbs carry 4 watts, and usually one burns out after each series of photographs. Bulbs of the same size carrying 6 watts were recently made for us.[1] These are probably more satisfactory, since they supply more light and each bulb can be used repeatedly, even though the battery is turned on to its full capacity.

From the Bronchoscopic Clinic of the Cedars of Lebanon Hospital.

1. These bulbs are made by the Electro-Surgical Instrument Co., Rochester, N. Y.

We use a De Vry 16 mm. motion picture camera with a standard lens mounting. We prefer it because: 1. Its shape and size conform admirably to our needs. 2. It has a side window which acts as a direct view finder. The image, therefore, can be visualized while one is photographing. This is more difficult in cameras with the view finder on top and at the front of the camera. 3. It can be set at various speeds. Any camera incorporating these features will, however, prove satisfactory.

A certain revision in mounting the lens is necessary. To secure proper focus and magnification it is imperative that the lens be moved forward from its usual position. We have accomplished this by having a collar, or band, made at a machine shop. This band is shown in figure 1 *A*. This is threaded at each end. It is screwed into the lens mounting, and into its distal end is screwed the lens itself. The band is of such a width as to advance the lens ¼ inch (0.6 cm.) from its usual position.

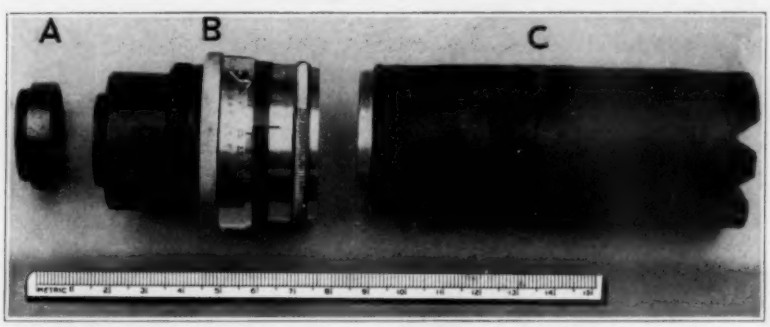

Fig. 1.—The adapter band (*A*), the lens (*B*) and the cardboard tube (*C*) used in taking motion pictures of the larynx.

The lens itself (fig. 1 *B*) is a standard 2 inch (5 cm.) lens opened to f/1.5. This arrangement of the lens results in a sharp focus at 11½ inches (29.2 cm.) and a usable focus between 11 and 12½ inches (27.9 and 31.7 cm.), measured from the front of the lens.

To maintain the camera at this exact distance from the vocal cords we have had built a cardboard tube (fig. 1 *C*) with a threaded collar at one end which screws onto the front of the lens. This tube is notched at its distal end to fit over the light carrier of the direct laryngoscope. Its diameter is exactly that of the 2 inch (5 cm.) lens. Bearing in mind that a direct laryngoscope is 7¼ inches (18.3 cm.) long and that we desire to maintain our camera at a distance of 11½ inches (29.2 cm.) from the vocal cords, it is obvious that this tube must represent the difference between these two lengths, which is 4¼ inches (10.7 cm.).

The distal end of the cardboard tube is notched superiorly, and when it is brought in contact with the laryngoscope this notch fits over the light carrier.

The camera and its rearrangement for our purpose are shown in figure 2 and are diagrammatically represented in figure 3.

These measurements must of necessity be varied in individual set-ups. For instance, if the width of the lens is not such as we have

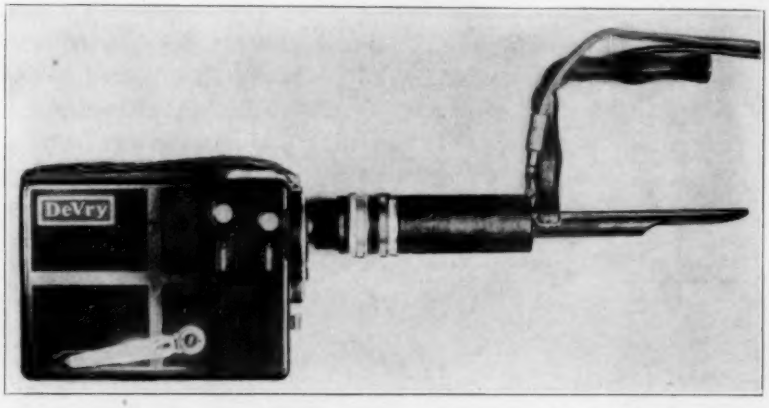

Fig. 2.—The camera and the laryngoscope arranged for taking pictures.

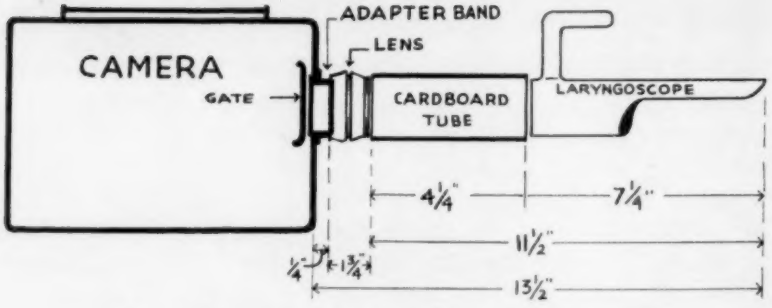

Fig. 3.—Diagram of the apparatus set up for use.

described, the adapter band must be made wider or narrower to compensate for this. It is important to make sure that an object placed 11½ or 12 inches (29 to 31 cm.) from the lens is in perfect focus. This can be tested by sighting on a piece of paper containing printed matter.

The speed of the camera is set at sixteen frames when we use the new 6 watt laryngoscopic bulb and at twelve frames with the old 4 watt

bulbs. We have had our best results in black and white with panchromatic negative film extra sensitive to red rays.

To this point we have outlined the camera set-up. Immediately prior to photographing (but after the camera has been set up) we warm the lens by wrapping a hot water bottle around it. This added warmth prevents fogging of the lens from the patient's breath. It is, of course, removed before the camera is used.

We are now ready to photograph the larynx with the expectancy of obtaining results such as those shown in figure 4. The projected motion pictures are more clearly defined than these examples, which are enlargements from single frames of the motion picture negative.

The larynx is exposed by the direct laryngoscope, as for any examination. When the laryngoscope is in place, the surgeon steps to one side, and the photographer points the camera down the tube of the

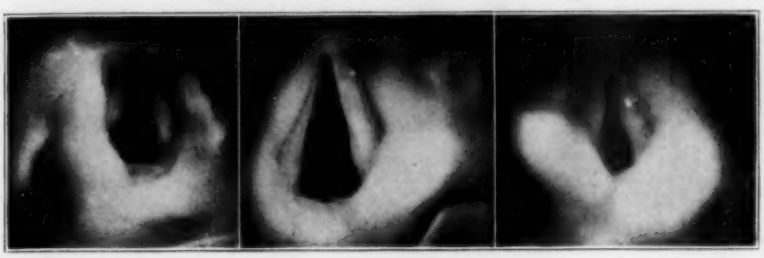

Fig. 4.—Enlargements from single frames of the motion picture negative.

instrument with the notch in the cardboard tube overriding the light carrier of the laryngoscope and sets the mechanism of the camera in motion. Success depends solely on the ability of the laryngologist to expose the larynx nicely and to hold the laryngoscope without motion and the ability of the photographer to aline the camera properly and to hold it still while photographing.

In a darkened room the photographer can visualize the image through the finder of the camera, but it is not absolutely necessary to do this, since with some experience proper alinement between the camera and the laryngoscope can usually be maintained. Proper alinement, of course, consists of holding the larynx, the laryngoscope and the camera in exact planes in all dimensions.

We have been able to obtain satisfactory color photographs by using the new Kodachrome A film for artificial light and building into the laryngoscope a second light carrier similarly placed but on the

opposite side of the laryngoscope, as illustrated in figure 5. This doubles the intensity of light.

Without question the method requires practice and a high degree of cooperation on the part of all concerned. One should expect nothing but failure at first. Good results, however, will come when proper alinement is achieved. It should be remembered that unless the larynx

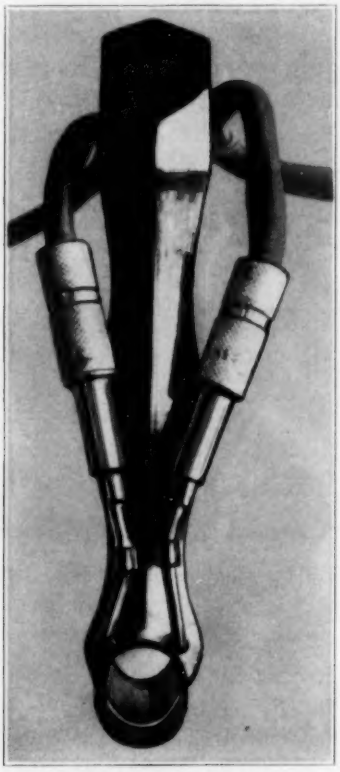

Fig. 5.—The laryngoscope with a second light carrier.

can be properly exposed with the laryngoscope, it cannot be photographed. Experience is best gained by practicing on patients accustomed to the passage of a bronchoscope.

As a result of these photographic studies, we have already learned a great deal about laryngeal physiology. These observations will be reported later.

No discussion of the problem of laryngeal photography is complete without reference to the excellent work of Dr. Francis E. Le Jeune, of New Orleans, who stands preeminent as a pioneer in this field.

SALIVARY CALCULUS CONTAINING A FOREIGN BODY

REPORT OF A CASE

JOHN A. PILCHER Jr., M.D.
ROANOKE, VA.

This case is reported, first, because the condition is apparently unique and, second, to show the importance of taking into consideration the patient's story, no matter how absurd it may seem. The lesion was an abscess of the submaxillary salivary gland of which the history was almost unbelievable until the findings proved it to be true.

The patient, a white man 28 years of age, came to the office on May 8, 1937, complaining of having had a sore throat for about two weeks and of being able to swallow only liquids. He was holding his head to one side, and he appeared to have a great deal of pain.

His history was interesting. About twelve years before he was examined, while chewing a piece of grass, he felt something stick into the soft tissue under the left side of his tongue. Since this accident there had been present more or less constantly a painful area under this side of his tongue, which became more disagreeable on eating. About six years before he was examined, a painful abscess formed in this area, which ruptured spontaneously within about five days, giving him relief. This area drained foul pus for about a week or ten days. Since the healing of this abscess there had always appeared a somewhat painful swelling under the tongue when he ate savory foods. This subsided within about one-half hour, with relief. He had sought relief from several physicians, relating to them the story of having stuck a piece of grass under his tongue. None of them placed credence in his story, and they treated him for an ordinary infection of the cervical glands, giving him general and local medication. About two weeks before I examined him (twelve years after the accident), the area under the left side of his tongue became swollen and painful after he ate pickles. The swelling failed to subside and became progressively greater and more painful, and he was unable to eat any solid food. The sight or odor of food increased the intensity of the pain. The swelling had become so painful that it was difficult for him even to attempt to open his mouth.

On examination there was noted a tender, somewhat fluctuant mass about 1½ inches (3.7 cm.) in diameter in the left submaxillary area just anterior and medial to the angle of the jaw. The patient could open his mouth but little and then with much pain and difficulty. The tongue and the floor of the mouth on the left side were elevated. The tonsils, teeth and gums showed no evidence of infection. In the floor of the mouth just medial to the left lower second molar was a pale mass about ¾ inch (1.8 cm.) long, which gave the impression of being the pointing of an abscess. Pressure on this mass caused pus to exude from the orifice of the duct of the left submaxillary salivary gland. No doubt there was

From the Gill Memorial Eye, Ear and Throat Hospital.

an abscess in or about the gland which opened into Wharton's duct. The history was more or less typical of partial blockage of the salivary duct and suggestive of a stone in the duct.

A roentgenogram of the left submaxillary area showed a shadow in the soft tissue at the location of the lower second molar. This shadow is shown in figure 1 and is suggestive of a stone in the submaxillary gland, apparently in or near the main duct.

Fig. 1.—Roentgenogram showing the shadow of the calculus just below the second molar. The picture was taken at an angle to show clearly the left submaxillary area.

Fig. 2.—The picture of the calculus is enlarged five times. Note the piece of grass passing through the upper portion of the calculus and the marked irregularity of contour of the calculus.

An incision about ¾ inch (1.8 cm.) long was made in the floor of the mouth parallel to the teeth at a location just above the shadow shown on the roentgenogram. An abscess cavity was encountered, from which was expressed about 2 drachms (7 cc.) of thick yellow pus. When the abscess cavity was probed, a hard stonelike mass was felt. By means of a blunt ring curet aided by external pressure on the submaxillary gland, a stone containing a piece of grass was dislodged and expressed. The stone, measuring 3 by 5 mm., surrounding a piece of grass 6 mm. long is shown enlarged five times in figure 2.

After the opening of the abscess and the removal of the stone the patient promptly got relief. He was able to open his mouth and to drink with almost no discomfort. The incision healed without complications, and he has remained symptom free. A communication from him two months after the operation stated that all discomfort disappeared about one week after the removal of the stone and that there had been no recurrence of symptoms.

CONCLUSIONS

I have been informed by the research department of the W. F. Prior Company, of Hagerstown, Md., that in a thorough review of the medical literature they have been unable to find any record of a case of formation of a stone about a foreign body in a salivary gland. Therefore, to the best of my knowledge, this is the first case of this condition to be reported in the medical literature.

INTRADURAL CONDITIONS IN RELATION TO RHINOLOGY AND OTOLOGY

A CRITICAL SURVEY OF RECENT LITERATURE

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

MISCELLANEOUS OTOLOGIC AND RHINOGENIC CONDITIONS

OLFACTORY SYSTEM

Physiology of Olfactory System.—The sense of smell is of vital importance in the lives of most of the members of the animal kingdom, for it is primarily connected with the search for food. Seydell [1] reviewed this function in some of the lower animals:

Coelenterata: Water hydra react to mechanical and chemical stimuli brought into contact with their tentacles, causing movements of the tentacles and the mouth slits.

Vermes: Worms also have an acute sense of smell. Only the head is sensitive to odors.

Insects: The olfactory end-organs in insects are probably located in the antennae; insects themselves produce odors for a variety of purposes.

Butterflies: These have the enticing odor developed to its highest degree.

Ants: Each ant hill, or colony, has its individual odor. If an ant from one hill is placed in another, it will be destroyed immediately, but if the antennae of the ants in this hill are removed this will not occur.

Bees: These can smell all odors which man is capable of sensing.

Fish: Fish differ from land animals in the structure of their olfactory end-organs, in the olfactory mucous membrane and also in the chemical composition of their plasma colloids and lipoids. In fish the odorivector is soluble in water and is brought to the olfactory apparatus in this medium. In many fish the sense of smell is acute.

Birds: These have an acute sense of smell.

Mammals: In the smaller land-grubbing animals smell is of the highest importance; the other senses are auxiliary to this sense. The

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A mimeographed index of all the articles surveyed by me for the ARCHIVES during the years 1925 to 1937 has been prepared, and a copy will be sent to any physician or library on personal application to Dr. W. P. Eagleton, 15 Lombardy St., Newark, N. J.

1. Seydell, Ernest M.: A Survey of Fact and Theory in the Feld of Olfaction, Ann. Otol., Rhin. & Laryng. **40**:472-508 (June) 1931.

mammals which live wholly or partly in the water, such as the dolphin, possess a rudimentary organ of smell; the olfactory mucous membrane is absent in the whale.

Seydell concluded that the chief olfactory center is in the uncus gyri hippocampi. He has found that the uncus and the other parts of the hippocampus are in close communication with each other. Brunner concluded that although the olfactory cortex of the macrosomatic animal has reached a greater quantitative development than that of man, it cannot be stated that the olfactory cortex in man has a merely rudimentary development from a qualitative angle.

All sense systems have primary and secondary sense centers. Lesions of the secondary sense centers of olfaction probably produce the so-called functional disturbances of smell. Man has a loss not of smell but of the ability to recognize given odors (agnosia). Lesions in the secondary olfactory centers produce agnosia. There is another form of disturbance of smell which results from lesions of the secondary olfactory centers, namely, hallucinations of smell. A number of cases of hallucinations of smell following injury to the brain have been recorded. In all cases of epilepsy with an aura of smell the cornu ammonis was found involved.

A person can express in words his conscious reactions to odors in three ways only: (1) by simile or metaphor, (2) by words denoting emotional response and (3) by words signifying a crude appreciation of the intensity of the stimulus.

The inability to describe a smell is not due to the limitation of the English language alone. The French poet Baudelaire showed an unusual preoccupation with odors and wrote some of his finest verse around them.

Parosmia from Metastatic Carcinoma of Olfactory Bulb.—A case of parosmia due to neoplastic involvement of the olfactory bulbs was recorded by Paskind.[2]

Three years after an operation for carcinoma of the breast a woman of 45 noted headache and dizziness and an unsteady gait, with a tendency to fall to the left. She complained of smelling an unpleasant odor and had left homonymous hemianopia. A carcinoma of the left cerebellar lobe was removed. At autopsy carcinomatous tissue was found in each olfactory bulb and invading the olfactory nerves.

Testing Olfactory Sense of Patients with Cerebral Tumor.—To investigate and measure the sense of smell Elsberg[3] devised a pro-

2. Paskind, Harry A.: Parosmia in Tumorous Involvement of the Olfactory Bulbs and Nerves, Arch. Neurol. & Psychiat. **33**:835-838 (April) 1935.

3. Elsberg, Charles A.: (*a*) The Sense of Smell: Value of Quantitative Olfactory Tests for the Localization of Supratentorial Tumors of the Brain, Bull. Neurol. Inst., New York **4**:511-522 (Dec.) 1935; (*b*) abstr., Arch. Neurol. & Psychiat. **36**:665-671 (Sept.) 1936; (*c*) The Newer Aspects of Olfactory Physiology and Their Diagnostic Applications, ibid. **37**:223-234 (Feb.) 1937.

cedure of "blast injection" or "stream injection" which makes it possible for a subject to perceive an odor even though he is holding his breath.

The apparatus consists of a set of nosepieces and test bottles. A known volume of the odor can be injected into one or into both nasal passages at a known pressure. The smallest quantity of a substance that when injected into one nasal passage can be identified by the subject is called the minimum identifiable odor, which is abbreviated as M.I.O. The acuity of monorhinal smell (injection into one nasal passage only) is thus determined, and the acuity of birhinal smell is likewise easily obtained. The M.I.O. on bisynchronorhinal injection is called the olfactory coefficient of the substance. Elsberg found that the efficiency of the olfactory receptors, though sensitive to certain other influences, is about the same in all normal persons.

By use of his apparatus Elsberg was able to reach certain conclusions with reference to quantitative olfactory testing. These were:

1. When a neoplasm exerts pressure on one olfactory bulb or tract, a larger quantity of the odorous substance must be injected into the ipsilateral nasal passage before the odor can be identified; that is, the M.I.O. is higher than normal on the side where a tumor is pressing on the olfactory peripheral apparatus.

2. If a tumor involves both olfactory bulbs or nerves, the M.I.O. is higher than normal on both sides but the greater increase is on the side more severely affected by the pressure.

3. Diminution of olfactory acuity occurs with an expanding lesion of the under-surface of the frontal lobe.

4. Diminution of olfactory acuity occurs in cases of suprasellar meningioma, aneurysm of the internal carotid artery, aneurysm of the circle of Willis or adenoma of the pituitary gland in which the growth projects above the sella turcica.

5. The duration of olfactory fatigue is prolonged on the side of the lesion, but the M.I.O. is normal in cases of intracerebral tumor or in the presence of a large dural growth that has become buried in the brain.

6. The duration of olfactory fatigue is prolonged to more than ten minutes in the presence of a tumor in or near the midline of the cranial cavity. For example, a parasagittal meningioma, a tumor of the corpus callosum or an infiltrating neoplasm which extends to the medial surface of one of the hemispheres.

7. The M.I.O. is higher on the affected side in cases of tumor of the frontal lobe when the growth extends to the under-surface of the lobe.

8. The M.I.O. may be less than normal in cases of generalized increase in intracranial pressure because of aggravated irritability of the olfactory pathways.

9. Localization of a tumor of the brain may be aided by analyzing the effects of olfactory fatigue on one side of the nose in birhinal smell or by studying bilateral fatigue in monorhinal smell.

10. Analysis of the effect of odors on the trigeminal nerve may help differentiate a cerebellar growth from a tumor of the lateral recess and may assist in the localization of tumor of the posterior fossa.

11. In cases requiring differentiation between tumor of the posterior fossa and tumor under the frontal lobe, an abnormally high M.I.O. will point to the latter, or, in any event, indicate that the neoplasm is not cerebellar.

These findings suggested to Tilney [4] that the part of the brain which is used for perceiving smell is probably larger than has been thought heretofore or that it is much more sensitive. He expressed the belief that this work would indicate that the function of the hippocampus cannot be limited solely to the perception or transmission of olfactory impulses. There is also ample reason to believe that the paleocortex has, or at one time did have, some somatic function. Perhaps the entity which has been identified as the "rhinencephalon" has no real existence, and it may be that smell is more diffusely represented in the brain than was previously thought.

Invasion of the Body Through the Olfactory Mucosa.—Rake [5] has observed that particles of prussian blue pass rapidly from the surface of the olfactory mucosa and within two minutes are found in the tissue spaces, in blood and lymph vessels, in the perineural spaces and in the pia-arachnoid membrane. Preliminary treatment of the olfactory mucosa with tannic acid causes inflammation of the mucosa and appears to prevent the pigment from entering the olfactory sensory cells. Pneumococci pass through the olfactory mucosa and reach the tissue spaces, the vessels and the subarachnoid space with the same rapidity as the pigment. They invade by passage between the cells of the mucosa. There is no apparent affinity of the organisms for the olfactory sensory cells.

THE NOSE IN POLIOMYELITIS, POLIOENCEPHALOMYELITIS AND EXPERIMENTAL VIRUS ENCEPHALITIS

Olfactory Tract as a Route of Infection in Poliomyelitis.—Both epidemiologic and experimental evidence suggest that the virus of

4. Tilney, F., in discussion on Elsberg.[3b]

5. Rake, G.: Rapid Invasion of Body Through Olfactory Mucosa, J. Exper. Med. **65**:303-316 (Feb.) 1937.

poliomyelitis reaches the nervous system through the nasopharynx and the olfactory nerves. The first nerve fibers are derived from cells which primordially are situated on the surface of the body, in the roof of the nasal cavity; the fibers penetrate the cribriform plate, reaching the olfactory bulb on the under-surface of the frontal lobe. Thus, the entire peripheral mechanism of the first nerve is exposed to the external environment, which perhaps accounts for the observations of Flexner and Clarke that the olfactory lobes become infected within forty-eight hours after intranasal inoculation. In the disease itself, the virus of poliomyelitis has been found in washings from the nasopharynx. The importance of the olfactory channel for this infection is indicated in the experiments of Schultz and Gebhardt,[6] who were able to produce the disease by intranasal inoculation and to prevent it by cautery of the olfactory bulb or section of the olfactory tract. Similar results were reported by Armstrong and Harrison,[7] who were able to reduce the incidence and mortality of poliomyelitis by instilling 1.5 cc. of a 4 per cent solution of alum into the nostrils of inoculated monkeys.[8]

Chemical Protection of Olfactory Tract in Poliomyelitis.—Schultz and Gebhardt[9] found zinc sulfate an efficient chemical agent for the protection of monkeys against poliomyelitis. They used a 1 per cent solution applied to the nasal mucosa of the infected animal. Of 53 monkeys previously inoculated with the virus of poliomyelitis, all but 2 survived; and of 22 monkeys inoculated with the virus and not treated with zinc sulfate, all but 2 died.

Since in rhesus monkeys the virus of poliomyelitis which enters the central nervous system by the olfactory nerves produces characteristic lesions in the olfactory bulbs, Sabin and Olitsky[10] suggested that postmortem examination of the olfactory bulbs may reveal the portal by which the virus has entered.

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6. Schultz, E. W., and Gebhardt, L. P.: Olfactory Tract and Poliomyelitis, Proc. Soc. Exper. Biol. & Med. **31**:728-730 (March) 1934.

7. Armstrong, Charles, and Harrison, W. T.: Prevention of Intranasally-Inoculated Poliomyelitis, Pub. Health Rep. **50**:725-730 (May 31) 1935.

8. The Olfactory Tract and Experimental Poliomyelitis, editorial, J. A. M. A. **105**:1986-1987 (Dec. 14) 1935.

9. Schultz, E. W., and Gebhardt, L. P.: Zinc Sulphate as a Chemoprophylactic Agent in Experimental Poliomyelitis, Proc. Soc. Exper. Biol. & Med. **35**:524-526 (Jan.) 1937.

10. Sabin, A. B., and Olitsky, P. K.: The Olfactory Bulbs in Experimental Poliomyelitis, J. A. M. A. **108**:21-24 (Jan. 2) 1937; abstr., Arch. Path. **23**:589 (April) 1937.

Flexner [11] found evidence that the virus of poliomyelitis ascends from the nasal membranes to the olfactory lobes of the brain and then, by direct nerve conduction, to the midbrain and the spinal cord. None of the virus passes from the nerve tissue directly into the cerebrospinal fluid. The cellular changes in the fluid represent a reaction of the nervous system to injury and not a transmission of the virus itself into the fluid.

Polioencephalomyelitis.—That encephalospinal poliomyelitis (polioencephalomyelitis) is a distinct clinical entity seems demonstrated by an experimental case cited by Brodie [12] in which a monkey intracerebrally inoculated with the virus of poliomyelitis showed encephalitic as well as spinal symptoms. Clinically, the animal gave symptoms of acute meningoencephalitis subsequent to the evidence of implication of the spinal cord. Autopsy showed characteristic encephalitic lesions in the brain, with infiltration of polymorphonuclear leukocytes, as well as the typical changes of poliomyelitis in the spinal cord. The cerebral condition was not secondary edema but definite encephalitic infection. While polioencephalomyelitis thus exists, it must be rare, as this was the only case of it in a series of 200 poliomyelitic monkeys.

Roentgen Treatment for Encephalitis from Nasally Injected Virus.—Since perivascular lymphocytic infiltration is the essential lesion of encephalitis and since lymphocytes are highly sensitive to irradiation, it seemed logical to Goldberg, Brodie and Stanley [13] to expect roentgen rays to be an adequate therapeutic measure in this condition.

They inoculated 91 mice intranasally with the virus of the St. Louis form of encephalitis (encephalitis B). Thirty-eight were treated with roentgen rays, and 53 were not treated. Roentgen treatments were given daily. Of the 53 untreated animals, all but 1 died (a mortality rate of 98 per cent). Of the 38 treated animals, 19 survived and 19 succumbed (a mortality rate of 50 per cent).

The authors' conclusion is that numerous roentgen treatments either prevent or cure encephalitis in mice inoculated intranasally with the virus.

RHINORRHEA

The commonest causes of cerebrospinal rhinorrhea are trauma and intracranial tumor. Diagnosis is established by identifying the discharge as spinal fluid. The specific gravity should be between 1.005 and 1.010, and when the fluid is boiled it should give no more than a

11. Flexner, Simon: Early Changes in the Cerebrospinal Fluid of Monkeys Nasally Instilled with the Virus of Poliomyelitis, Science **78**:129-130 (Aug. 11) 1933.

12. Brodie, Maurice: Cerebral Involvement in Acute Anterior Poliomyelitis, Am. J. Dis. Child. **48**:57-68 (July) 1934.

13. Goldberg, Samuel A.; Brodie, Maurice, and Stanley, Phyllis: Effect of X-Ray on Experimental Encephalitis in Mice Inoculated with the St. Louis Strain, Proc. Soc. Exper. Biol. & Med. **32**:587-590 (Jan.) 1935.

trace of globulin coagulum. In a case recorded by Learmonth,[14] he did a transfrontal flap operation, packed the cribriform plate with iodine packs (to encourage scarification) and subsequently gradually removed the packs. The rhinorrhea appeared to be cured. Smith and Walter [15] reported a case of cerebrospinal rhinorrhea associated with a pituitary cyst.

Their patient, a man of 51, had for two months complained of a daily discharge of watery fluid from the left nostril. He suffered from pain over the eyes, neck and left side of the head, and a diagnosis of sinus disease had been made. His temperature was 101.9 F. The nasal flow was intermittent and irregular in quantity. The leukocyte count, which was 26,500 on admission, dropped to 6,300 within two days. The fluid gave a 2 plus reaction to tests for albumin and for globulin. It contained a few lymphocytes and many pneumococci. Roentgenograms disclosed calcium absorption of the clinoid processes and the sellar floor. Preliminary elevation of a flap of the frontal bone exposed a bulging area. After the pressure was reduced closure was effected. This stopped the rhinorrhea. A week later a cystic growth in the pituitary body was punctured and fulgurated. Recovery was prompt.

The fever and leukocytosis were probably due to localized meningitis. The increased intracranial pressure could scarcely have accounted for the fever and certainly did not cause the leukocytosis. Although the fistulous tract was not affirmatively located, it seemed probable that there was an opening into the sphenoid sinus, which when filled emptied normally into the nose through the natural opening between that sinus and the nasal cavity.

Rhinorrhea Cured by Plugging Enlarged Openings in Cribriform Plate with Wax.—Graham's [16] patient had a history of attacks of vomiting since childhood, varying in frequency. She had a constant watery discharge from the left side of the nose for five months. She had tinnitus occasionally in the left ear. She had become very stout during the years preceding the onset of the nasal discharge.

The roentgenogram disclosed almost complete destruction of the pituitary fossa; its floor was pressed downward, and the sphenoid sinuses were almost obliterated (which indicated increased intracranial pressure, probably from a suprasellar tumor).

A flap of the frontal bone was turned down. The cribriform plate was wide and contained unusually large perforations. When the dura

14. Learmonth, J. R.: Cerebrospinal Rhinorrhea Treated by Operation, Proc. Staff Meet., Mayo Clin. **4**:115 (April 10) 1929.

15. Smith, C., and Walter, L.: Cerebrospinal Rhinorrhea with Cyst of the Pituitary Body, Arch. Otolaryng. **14**:610-614 (Nov.) 1931.

16. Graham, T. O.: Cerebrospinal Rhinorrhoea, J. Laryng. & Otol. **7**:344-347 (May) 1937.

was reflected over the lesser wing of the sphenoid, cerebrospinal fluid flowed. The openings in the cribriform plate were filled with wax.

Eight months later the patient reported that there had been no rhinorrhea except an occasional drop from the left nostril.

Graham suggested that the pressure on the pituitary gland had induced the accumulation of fat, though she showed no other obvious signs of Fröhlich's syndrome—infantilism. dwarfism, Simmonds' disease or diabetes insipidus.

INTRACRANIAL COMPLICATIONS OF CRANIAL OSTEOMYELITIS

Osteomyelitis of the Frontal Bone.—Reviewing 91 cases in which osteomyelitis of the frontal bone was reported as a complication of sinus disease, Lemon [17] found that the average age of the patients was 25, two thirds of them being between 15 and 30 years old. Sixty per cent were male. The commonest organism was Staphylococcus aureus. In most of the cases the frontal sinus was the primary source. In 1.5 per cent the osteomyelitis followed acute maxillary sinusitis, and in 6 per cent it followed chronic maxillary infection. In 42 per cent of the cases the osteomyelitis complicated acute infection, and in 35 per cent it complicated chronic infection of the frontal sinuses. Seven of the 91 patients had had pansinusitis.

The death rate was 5.6 per cent; the osteomyelitis in 55 per cent of the fatal cases followed acute sinus infection; in the remainder it was associated with chronic sinusitis. In patients whose osteomyelitis followed an operation, the average interval between operation and onset was ten days. The average interval between the onset of the osteomyelitis and the termination of the condition (death or recovery) was thirteen months. In a third of the cases abscess of the brain was a complication.

It appeared that the infection traveled in the diploe between the tables of the skull; it was always more advanced than the roentgenographic findings suggested. The mortality was higher in postoperative cases, and the postoperative osteomyelitis was not as self-limited as that which occurred spontaneously.

Von Eicken [18] stated that a large frontal sinus is usually surrounded by well developed diploe, in its upper and lateral aspects. The diploetic veins of Breschet do not follow the suture lines with any exactness but are prone to be irregular in site and direction. Wischnowski has designated one of these veins the "vena norma temporalis."

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17. Lemon, A. Neil: Report of Case of Maxillary Sinusitis with Complicating, Spreading Osteomyelitis, Brain Abscess and Death, Laryngoscope **46**:754-764 (Oct.) 1936.

18. von Eicken, C.: Osteomyelitis of the Skull, Proc. Roy. Soc. Med. **29**: 192-196 (Dec.) 1935.

Watkyn-Thomas and McKenzie [19] expressed the belief that a staphylococcus is, as a rule, the organism responsible for osteomyelitis of the frontal bone. They presented bacteriologic findings in 1 case, however, in which, although Staph. aureus was cultured from the periphery of the suppurating portion, cultures of material taken from bone some distance in advance of the suppuration showed a few colonies of a "coliform bacillus" and an occasional streptococcus.

Blair and Brown [20] found a streptococcus in short chains in 1 case, a "streptococcus" in 2 cases, a streptococcus associated with a staphylococcus in 1 and an actinomyces in another.

Woodward [21] expressed the opinion that in all cases the condition is attributable to infection with Staphylococcus pyogenes-aureus.

Mosher and Judd [22] found Staph. aureus present in all their cases, and Mosher,[23] in a recent article, expressed himself as of the opinion that Staph. aureus is the causative organism.

Williams and Heilman [24] stated that they recognize three clinical types of osteomyelitis of the frontal bone secondary to disease of the frontal sinus: (*a*) the fulminating type, (*b*) the localizing type and (*c*) the spreading type. Of 13 cases observed by them, the condition in 4 was of the fulminating type, and all 4 patients died, meningitis developing in from twelve to twenty-four hours after the first symptom of sinusitis.

The fulminating type frequently follows swimming and is characterized by intense frontal pain and generalized headache, high fever, rapidly spreading edema and a tendency to invade the arachnoid and the cranial sinuses.

In an effort to resolve the divergent opinions regarding the organism which causes this disease Williams and Heilman [24] reported a case.

The patient previously had undergone intranasal operations. A submucous resection was performed, and a week later external frontoethmosphenoid sinusostomy on the right side was done. Three weeks later a similar procedure was undertaken on the left side.

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19. Watkyn-Thomas, F. W., and McKenzie, Dan: Bacteriology of Spreading Osteomyelitis, J. Laryng. & Otol. **44**:253-254 (April) 1929.

20. Blair, V. P., and Brown, J. P.: Septic Osteomyelitis of the Bones of the Skull and Face, Ann. Surg. **85**:1-26 (Jan.) 1927.

21. Woodward, F. D.: Osteomyelitis of the Skull, J. A. M. A. **95**:927-929 (Sept. 27) 1930.

22. Mosher, H. P., and Judd, D. K.: Seven Cases of Osteomyelitis of the Frontal Bone, Laryngoscope **43**:153-212 (March) 1933.

23. Mosher, H. P.: Osteomyelitis of the Frontal Bone, J. A. M. A. **107**:942-946 (Sept. 19) 1936.

24. Williams, Henry L., and Heilman, Fordyce R.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus, Arch. Otolaryng. **25**:196-207 (Feb.) 1937.

The patient's condition was satisfactory until the thirteenth day, when a swelling developed over the left brow. A month later the lesion was still expanding and showed a tendency to extend toward the temporal fossa.

Because of the report of Watkyn-Thomas and McKenzie,[19] material for culture was taken from the interior of the sequestrum.

This showed an anaerobic streptococcus. The preparation of an antivirus was advised. Through and through lavage of the wound was carried out with 2 cc. of Rosenow's concentrated hyperimmune polyvalent antistreptococcus serum placed in 50 cc. of a sterile physiologic solution of sodium chloride. The response seemed definite.

They summarized: 1. The finding of the identical organism, an anaerobic streptococcus, in 2 cases of osteomyelitis of the frontal bone, together with the apparently unusually favorable results obtained by specific therapy with an autogenous antivirus, suggests that the organism causative of this lesion may have been isolated. 2. The conjecture that the disease is of staphylococcic origin has not seemed satisfactory, because staphylococci frequently are present in sinuses which have been operated on without producing invasive osteomyelitis. 3. Treatment with the antivirus clinically seemed to have a favorable effect in their 2 cases. They expressed the belief that in cases of fulminating osteomyelitis, in which meningitis and sepsis often appear in from twelve to twenty-four hours after the first appearance of symptoms, radical surgical operation tends to hasten the spread of the disease and only enough operating should be done to relieve the pressure and drain the pus in the frontal sinus. In consideration of the fact that Furstenberg [25] and Mosher [23] have demonstrated that propagation of the disease is by thrombosis of the dural veins communicating with the dural sinuses and the intradural veins, it would seem best to eradicate the primary disease in the bone before attempting to treat complications, such as suppurative encephalitis or thrombosis of the dural sinuses.

Inflammation and Thrombophlebitis of Sagittal Sinus and Subdural Abscess of Nasal Origin.—A case of fatal suppuration in the subdural space following nasal sinusitis was presented by Macgregor and Smith.[26]

An 8 year old girl had had a "cold in the head." She had severe headache, nasal catarrh, fever with a temperature of 101 F. and marked swelling of the upper lid. The movements of the eyeball were restricted. Pus was found below the orbital periosteum in the supra-orbital region, having come through a fistula in the floor of the frontal sinus. Four days later she had right hemiplegia and aphasia (which was thought to be due to a metastatic abscess in the left cerebral hemisphere). The left parietal bone was trephined. Pus exuded under pressure

25. Furstenberg, A. C.: Osteomyelitis of the Skull, Ann. Otol., Rhin. & Laryng. **40**:996-1012 (Dec.) 1931.

26. Macgregor, A. R., and Smith, A. B.: A Case of Sagittal Sinus Thrombosis and Subdural Abscess Following Nasal Sinusitis, J. Laryng. & Otol. **50**:600-603 (Aug.) 1935.

from the medulla of the bone and from the dural surface. The dura was not incised. Nonhemolytic streptococci were cultured from the pus; similar organisms were found in cultures of blood. The spinal fluid was sterile.

Postmortem examination revealed pus in the sphenoid and ethmoid sinuses on both sides. Two osteomyelitic foci were seen in the left parietal bone. There was an abscess in the left subdural space, the pus being in the largest amount in the upper anterior part of the parietal lobe, spreading forward to the frontal pole and passing down the medial surface of the hemisphere and the lower surface of the temporal and occipital lobes to reach the floor of the middle cranial fossa.

A septic clot filled the entire length of the superior sagittal sinus. Septic thrombi were also found in the meningeal veins tributary to the sagittal sinus on the left side, draining the area of the subdural abscess.

Turner and Reynolds [27] quoted Burger as reporting that in 83 per cent of his cases of thrombosis of the sagittal sinus (23 of 28 cases) the process originated in the frontal sinus.

THROMBOSIS OF CAVERNOUS SINUS

Hachem [28] distinguished between simple thrombophlebitis and suppurative thrombophlebitis of the cavernous sinus. The former is a "relatively benign affection," productive and constructive in its pathologic process, which results in a compact thrombus adherent to an indurated sinus wall. Hachem found a mortality rate of from 95 to 93 per cent, but cited my work,[29] in which 3 cures were effected in 24 cases (a mortality rate of 87 per cent) by surgical treatment. He recommended the use of the thermocautery for the opening of facial or nasal pustules.

In a review of 12 cases of thrombosis of the cavernous sinus Goldman [30] found 4 in which the process originated in the nasal accessory sinuses. The ethmoid sinuses alone were the source in 2 of these, the ethmoid and maxillary sinuses in 1 and the ethmoid and sphenoid sinuses in 1. In the last-mentioned case the condition was associated with osteomyelitis.

A case of thrombosis of the cavernous sinus was assigned by Hector and Bozorth [31] to dental infection, but which they stated might have progressed through venous channels in the alveolar bone and thence to

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27. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases, Edinburgh, Oliver & Boyd, 1931.

28. Hachem, Ismail: Clinical, Anatomic and Pathologic Studies of the Orbital Infections Designated Under the Term of Cavernous Sinus Thrombophlebitis, Rev. d'oto-neuro-opht. **11**:65-67 (Jan.) 1933.

29. Eagleton, Wells P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1927.

30. Goldman, J. L.: Cavernous Sinus Thrombosis Following Intranasal Infections, J. Mt. Sinai Hosp. **2**:121-125 (Sept.-Oct.) 1935.

31. Hector, George A., and Bozorth, Squire: Cavernous Sinus Thrombophlebitis: Report of a Case, J. Am. Dent. A. **19**:1765-1767 (Oct.) 1932.

the pterygoid plexus and through the foramen Vesalii to the cavernous sinus. However, in my opinion the primary focus was osteomyelitis of the basis from an infected sphenoid sinus, brought into activity by surgical manipulation on the teeth, as she had initial tenderness and rigidity of the left side of the neck and pain on pressure over the cervical portion of the spine, which I have observed [31a] on numerous occasions and the segmental character of which I have pointed out.

A woman of 46 acquired pain in a right upper tooth, which radiated to the left side of the neck and head. There were a postnasal discharge, marked tenderness of the left side of the neck with rigidity and pain on rotation, and pain on pressure over the cervical portion of the spine and the muscles of the left side of the neck. It was felt that the cervical portion of the spine was affected, especially on the left side, either with an inflammatory disease of the bone or with synovitis of the upper cervical vertebrae.

The maxillary teeth were removed with the patient under nitrous oxide anesthesia. The next day the entire right eyeball was exophthalmic and its conjunctiva chemotic. Extra-ocular movements were restricted. The nose revealed pus formation with polypoid degeneration, and a hyperplastic membrane in the anterior wall of the sphenoid sinus. Partial exenteration of the anterior and posterior ethmoid cells was carried out. The patient died six hours later.

At autopsy the brain was found to be edematous. The right ethmoid sinuses were filled with blood clot and the sphenoid sinus with mucus. A purulent exudate covered the internal lining of the cavernous sinus, and abscesses were seen in the sinus on histologic study. In the maxillary sinuses the stroma was hyperemic and the epithelial lining was edematous. Only mild inflammatory changes were found in the sphenoid sinus. The lungs were studded with multiple abscesses and multiple bacterial emboli.

Too little attention is paid to dental foci from which thrombosis of the cavernous sinus may develop.

Morgenstern's [32] patient, a woman of 50, had a third left lower molar extracted. Shortly thereafter she had fever, chills and swelling of the cheek and lower eyelid and complained of severe headache and blurred vision. Roentgenograms suggested osteomyelitis of the left ramus of the jaw. She became stuporous and irrational. The jaw was drained; four days later she had bilateral exophthalmos and rigidity of the neck. The right eyeball was fixed and the pupil did not react to light. She died of pulmonary edema. At autopsy the bone between the left mandible and the middle cerebral fossa was found infiltrated with pus. Pus and necrotic areas were noted in both parts of the hypophysis, and the angle between the frontal and the temporal lobe showed a collection of pus in the leptomeninges. The left cavernous sinus contained a purulent clot. The left ophthalmic and the left jugular vein were thrombosed. Several specimens of bone from the region of the sinus on the left side and from the left lower jaw microscopically showed osteomyelitis. There was present throughout a toxic-degenerative index of from three to four plus, which indicated a serious prognosis.

31a. Eagleton, W. P.: Tr. Am. Laryng., Rhin. & Otol. Soc., 1937, to be published.

32. Morgenstern, Bernard P.: Cavernous Sinus Thrombosis of Dental Origin, Arch. Otolaryng. **21**:442-447 (April) 1935.

Bekir [33] reported a case of otitis media in which the veins of the bone transmitted the infection first into the petrosal sinus and subsequently into the cavernous sinuses.

For seven years the patient, aged 21, had had chronic suppurative otitis media. Mastoidectomy was done. Three weeks after operation headache developed, first in the temporoparietal area and then between the eyebrows. A week later tender exophthalmos on the right side was noted. Then the left eye began to bulge. The eyeballs were practically immovable. Autopsy revealed purulent thrombosis of the right superior and inferior petrosal sinuses and bilateral thrombosis of the cavernous sinus.

A case of thrombosis of the cavernous sinus in which recovery ensued without removal of the eyeball was reported by Jauerneck.[34] The infection was apparently of tonsillar origin.

The preservation of sight in the one (right) eye was possible because early operation on that side promptly relieved the tension in the orbit.

After a tonsillar infection a woman 34 years old exhibited proptosis of the left eyeball, with swelling of the lids. Jauerneck opened the ethmoid labyrinth and removed the lamina papyracea and the ethmoid cells. The orbital fat bulged into the opening. No pus was seen either in the ethmoid sinuses or in the orbit. After this procedure there was improvement on the left side (i. e., the one operated on), but exophthalmos appeared on the right side. To relieve tension in the right orbit Jauerneck entered through the maxillary sinus and removed the floor of the orbit. The patient recovered; the left eye remained blind.

It is known that arteritis may be associated with thrombosis of the cavernous sinus. In Neuschüler's [35] case it would appear that a nasal infection which extended into the cavernous sinus by the venous route produced basal leptomeningitis with arteritis that involved the adventitia and the media of the vessels. Erosion from the extensive periarteritis produced a hemorrhage into the ventricles.

After the development of a nasal furuncle a man of 30 suffered from pain in the left frontal area, with edema of the left upper eyelid and proptosis of the left eyeball. Kernig's sign was positive. Autopsy revealed thrombosis of the cavernous sinus complicated by bleeding into the lateral ventricle on the left side and into the third and fourth ventricles.

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33. Bekir, Vahdettin: A Case of Otogenic Cavernous Sinus Thrombosis, Ann. d'oto-laryng., June 1934, pp. 626-628.

34. Jauerneck, A.: Cavernous Sinus Thrombosis of Tonsillar Origin, with Recovery, Hals-, Nasen- u. Ohrenarzt **27**:271-273, 1936.

35. Neuschüler, I.: Thrombophlebitis of the Cavernous Sinus with the Rare Onset of a Ventricular Cerebral Hemorrhage, Riv. oto-neuro-oftal. **8**:301-316 (July-Aug.) 1931.

Venous stasis may mimic the picture of thrombosis of the cavernous sinus. Beyer [36] reported the case of a 21 year old woman who five days after a tooth had been extracted suffered from fever and exophthalmos —the classic symptom complex of thrombophlebitis of the cavernous sinus—yet autopsy revealed only stasis of blood in the entire right cerebral hemisphere.

Schumacher [37] recorded a case of slowly invasive thrombophlebitis involving successive venous radicles of the side of the skull opposite the original locus of the infection.

A 22 year old woman had a discharge from the left ear for years. A furuncle developed over the medial end of the left eyebrow. It apparently healed. Two days later she complained of headache and insomnia. Examination disclosed obliteration of the left nasolabial fold, weakness of the left abducens nerve and stiffness of the neck. After a radical mastoidectomy on the left side three days later, she had exophthalmos on the right side. A week later the right orbit was opened; no pus was found. Autopsy revealed purulent staphylococcic meningitis at the base of the brain, with a purulent clot filling the right cavernous sinus and suppuration in the right inferior petrosal sinus and in the right basilar plexus. Purulent infiltration had also invaded the capsule of the pituitary gland. Foci of pus were present in the lung.

A condition such as that presented by this patient should be amenable to surgical treatment if the site of the advancing infection is attacked early.

Cases of Cured Thrombosis of Cavernous Sinus.—Since the publication in 1927 of my book [29] entitled "Cavernous Sinus Thrombosis," Cavenagh [38] collected the 12 cases of recovery that have been reported in the literature. He divided them as to the type of treatment employed. In 3 the sinus itself was operated on. Four patients were successfully treated by antiinfectious therapy. In 1 case there was spontaneous recovery. In 4 the condition was chronic and was treated by direct operation on the sinus.

Browder [39] in 1933 had a patient who had thrombosis of the cavernous sinus after a carbuncle on the nose. The sinus was treated by electrocoagulation through an intracranial approach. The affected eye became permanently blind.

In Christophe's [40] case it was possible to preserve a functioning eye in spite of an orbital operation. After a toothache the patient acquired blindness, exoph-

36. Beyer, T. E.: The Syndrome of Cavernous Sinus Thrombosis Due to Venous Congestion of the Right Cerebral Hemisphere, Laryngoscope **42**:632-634 (Aug.) 1932.

37. Schumacher, O.: Facial Furuncle, Thrombosis of the Cavernous Sinus and Terminal Meningitis, Arch. f. klin. Chir. **169**:789-794, 1932.

38. Cavenagh, J. B.: Cavernous Sinus Thrombosis: A Study of the Cases of Recovery, Brit. M. J. **1**:1195-1199 (June 13) 1936.

39. Browder, E. J.: Carbuncle of Nose; Ophthalmic Vein Phlebitis; Operation for Cavernous Sinus Thrombosis; Recovery, Laryngoscope **43**:829-830, 1933.

40. Christophe, L.: Thrombophlebitis of Cavernous Sinus; Operation; Cure, J. de chir. et ann. Soc. belge de chir. **27**:312-318, 1928.

thalmos and limitation of movement of the eyeball. At operation Christophe followed the Eagleton technic. He clamped the common carotid artery and turned back an orbital osteoplastic flap. It was possible to incise and drain the cavernous sinus without removing the eyeball. The patient made a complete recovery.

Stout [41] in 1931 published a case of recovery from septic thrombosis of the cavernous sinus in which the blood culture was positive for Staph. aureus. Bacteriophage was given both intravenously and intrathecally. On the ninth day the right eye showed chemosis and proptosis. Daily intravenous injections of bacteriophage were continued until urticaria occurred. About the thirty-second day of illness severe pains in the head and neck and vomiting occurred. Fluid obtained by a spinal puncture showed a high cell count and staphylococci on culture. A trial intrathecal injection of 1 cc. of bacteriophage was made, and, as it was well tolerated, a daily injection of 4 cc. was given for four days. The patient was discharged on the seventy-eighth day of the illness.

Lewis [42] also used bacteriophage intravenously. His patient had apparently hopeless meningitis, with a temperature of 104 F., chemosis, proptosis and signs of involvement of the cranial nerves. Recovery was slow, but by the end of the third month the patient was well except for blindness in the affected eye. He later died, and the autopsy showed a healed cavernous sinus.

In Weizenhoffer's [43] case the infection of the cavernous sinus developed on the side opposite the original focus, which was a furuncle in the right auditory meatus. The left eye was chemotic and exophthalmic, and blood culture showed a staphylococcus. The only treatment was repeated transfusion of citrated blood. The patient made a complete recovery.

Gerrie's [44] patient acquired the classic signs of thrombophlebitis of the cavernous sinus after a submucous resection. A septal abscess was opened twelve days later, and the involvement of the cavernous sinus became apparent on the following day. Subsequent to the repeated intramuscular and intravenous injection of antistreptococcus serum, he made a complete recovery.

Cavenagh [38] added a case of his own in which the infection was practically self-limited, although some accessory surgical measures were adopted.

A woman aged 26 had been hard of hearing in the left ear for many years. She had fever and a discharge from that ear. A few weeks later the right eye was proptotic. A cell count showed 39,000 leukocytes. An abscess in the thyroid gland was evacuated. The ophthalmoscopic signs suggested thrombosis of the cavernous sinus. A clot was removed from the lateral sinus. The anterior triangle of the neck contained an abscess cavity extending to the base of the skull. The internal jugular vein was collapsed. This vein was ligated. Two weeks later the proptosis

41. Stout, B. F.: Septic Cavernous Sinus Thrombosis: Report of Two Cases with Recovery of One Following Bacteriophage Therapy, J. Lab. & Clin. Med. **17**:28-39, 1931.

42. Lewis, E. R.: Cavernous Sinus Thrombosis; Recovery, Ann. Otol., Rhin. & Laryng. **40**:341-347, 1931.

43. Weizenhoffer, A.: Contralateral Cavernous Sinus Thrombosis Following Furuncle of External Auditory Canal; Recovery, New York State J. Med. **32**: 139-142, 1932.

44. Gerrie, J.: Cavernous Sinus Thrombosis with Recovery, Brit. M. J. **2**: 453, 1935.

began to subside. A month later the eye was normal except for relative pallor of the left disk and slight contraction of the visual field.

Summarizing the therapeutic method employed in the 13 cases, Cavenagh suggested that a combination of (*a*) blood stream therapy, (*b*) operation on the sinus itself and (*c*) ligature of the internal or the common carotid artery offers the most promising results. He emphasized the importance of putting the inflamed sinus at rest by ligating the carotid artery, which I first suggested some years ago. Cavenagh also recalled my insistence on the importance of eradicating the primary focus and draining the route of invasion.

Of the 13 cases listed by Cavenagh, the thrombosis in 7 may be described as of the acute infective type.

Treatment by Electrical Coagulation of Cavernous Sinus.—A case of thrombophlebitis of the ophthalmic vein was reported by Browder and Myerson,[45] in which it was possible to prevent thrombosis of the cavernous sinus through electrical coagulation of the contents of the sinus without enucleating the eye.

A week after the appearance of a boil on the right side of the nose, the patient suffered from headache and chills and noted swelling of the lid and exophthalmos of the right eye. Ocular movements on the affected (right) side were restricted. The right fundus had papilledema on the nasal side. A positive Kernig sign was elicited. Staph. aureus was found both in the blood stream and in the pus from the nasal carbuncle.

Operation and Course: The dura was incised through a subtemporal opening, and the pole of the temporal lobe was retracted. The sphenoid ridge was exposed and followed to the tuberculum sellae, where the cavernous sinus was visualized. The sinus appeared normal. Its lumen was obliterated by first coagulating the surface and then introducing the needle into the anterior aspect of the vessel. The right angular vein was opened externally and found to contain pus, which on culture revealed Staph. aureus. Exophthalmos persisted for three weeks. Vision became progressively poorer in the right eye, and within a few days the patient became blind in that eye. During the first two weeks after the operation several incisions were made into the right orbit to evacuate pus. The blood culture remained positive for the staphylococcus until a fortnight after the operation, when it became sterile. Two days later the patient acquired multiple metastatic abscesses in the chest, which gradually healed.

It seems to me that from a surgical standpoint it would have been better to attack and drain the sinus from in front after removal of the eye.

The coagulation of the anterior end of the cavernous sinus was immediately followed by (*a*) increase of the chemosis and proptosis, (*b*) fixation of the eyeball and (*c*) loss of sight from opacity of the intra-ocular media (all due to the obliteration of ophthalmic veins by

45. Browder, Jefferson, and Myerson, Mervin C.: Surgical Method for the Prevention of Thrombophlebitis of the Cavernous Sinus, Arch. Otolaryng. **21**: 574-583 (May) 1935.

the coagulation). This was followed by low grade venous sepsis, as shown by the low temperature, constant sweating and positive blood culture (staphylococci), the end-result being the preservation of a blind, disfiguring eye.

Browder and Myerson's [45] method is original and is to be commended as an advance in the operative treatment of thrombophlebitis of the cavernous sinus, but it should not confuse one's thinking as to the necessity for direct drainage of septically thrombosed veins. Its greatest advantage, in my opinion, is that it will be permitted by a patient while he has a seeing eye, which has been the cause of refusal of an early operation in many of my cases.

A case of thrombosis of the cavernous sinus successfully treated by enucleation of the eye and drainage through the orbit was reported by Dupuy, Brewster and Thomas.[46]

After furunculosis of the upper lid and cellulitis in the left orbit, the patient had retinal hemorrhages, fever, stupor and exophthalmos. The operation consisted of ligation of the left internal carotid artery, enucleation of the left eye and exenteration of the orbital contents. Pus was encountered behind Tenon's capsule. The bony lamina between the optic foramen and the sphenoid fissure was removed. The sinus, which was found blocked by a thrombus, was opened and drained. Although the patient had bacteremia, with the culture of blood positive for Staph. aureus, he recovered. Gentian violet and mercurochrome were administered intravenously, and antistreptococcus and erysipelas antitoxins were employed.

I [47] first expressed the idea that the pulsation of the internal carotid artery as it courses through the cavernous sinus is a factor in spreading the infection intracranially. The arterial pounding against the thrombus causes the infection to advance through the circular veins and promotes extension into the opposite sinus.

TRAUMATISM OF OLFACTORY APPARATUS

Olfactory Disturbances from Head Injury.—Helsmoortel, Nyssen and Thienpont [48] studied the sense of smell in 43 persons who had had injury to the head. Both pure olfactory stimuli and substances which affected the fifth and ninth cranial nerves as well as the first were used. The degree of anosmia was gaged by the time elapsing between the presentation and the perception of the odor. Of the 43 cases, there

46. Dupuy, Homer; Brewster, H. F., and Thomas, Ashton: Left Cavernous Sinus Thrombosis, Laryngoscope **42**:931-933 (Dec.) 1932.

47. Eagleton, Wells P.: The Carotid Venous Plexus as the Path of Infection in Thrombophlebitis of the Cavernous Sinus, Arch. Surg. **15**:275-287 (Aug.) 1927.

48. Helsmoortel, J.; Nyssen, R., and Thienpont, R.: Olfactory Disturbances in Patients with Cranio-Cerebral Traumatism, Rev. d'oto-neuro-opht. **11**:489-502 (July) 1933.

were (*a*) 6 of complete and lasting anosmia, (*b*) 2 of anosmia evolving into hyposmia, (*c*) 7 of partial or variable anosmia, (*d*) 3 of olfactory disturbances due to mental torpor, (*e*) 2 of anosmia due to a preexisting nasal lesion and (*f*) 1 of simulated anosmia. Of the 6 patients with complete and lasting anosmia, 5 showed fracture, and 3 of these had fracture involving the ethmoid bone. On the other hand, of the 9 patients presenting less severe olfactory disturbances, a fracture was found in only 1.

Fractures Through Frontal Sinus.—Among 125 cases of fracture of the frontal sinus Gurdjian and Shawan [49] found 71 in which there was no bleeding from the nose. In 52 bleeding occurred for only a day or two after the trauma. In the other 2 cases there was continuous oozing of blood and spinal fluid, lasting for one day in 1 case and for two days in the other.

Of the 2 patients with spinal fluid rhinorrhea, 1 recovered without operative intervention. The other died, although there was no evidence of meningitis. Cerebrospinal fluid rhinorrhea must be considered a serious complication of fracture of the anterior fossa; in Campbell's [50] series the incidence of meningitis was high, although some patients recovered without surgical treatment.

Of the 125 patients of Gurdjian and Shawan, 9 died and 116 recovered. Three had compound fractures. Thirty-eight showed extension of the fracture toward the base of the skull. In 3 cases cloudiness in the sinus suggested intrasinus bleeding. Only 1 died from meningitis—a patient with bleeding from the nose without cerebrospinal rhinorrhea. When meningitis develops (in this series in less than 1 per cent of the cases), it is probably associated with involvement of the emissary veins at the base. In the presence of suppurative sinusitis the face-down position may afford gravity drainage and may curb extension of the infection toward the cerebrum.

Gurdjian and Shawan summarized their observations: 1. The incidence of fracture of the frontal sinus in a large series of cases of fracture of the skull was about 5 per cent. 2. Patients with fracture of the frontal sinus should be confined to bed for a week or ten days at least; they should be forbidden to blow the nose, and intranasal douches should not be used. 3. Although the incidence of meningitis was only 0.8 per cent in their series, infection in the frontal sinus should be watched for. 4. Conservative treatment is generally advisable, but surrounding cellulitis and evidences of infection call for surgical

49. Gurdjian, Elisha S., and Shawan, H. K.: Management of Skull Fractures Involving the Frontal Sinus, Ann. Surg. **95**:27-32 (Jan.) 1932.

50. Campbell, E. H.: Cerebrospinal Rhinorrhea Following Intracranial Surgery, Ann. [illegible] Rhin. & Laryng. **37**:865-876 (Sept.) 1928.

intervention. 5. A compound fracture of the frontal sinus should be operated on as soon as the condition of the patient permits. At operation the posterior wall of the sinus should be inspected.

It seems to me that the authors entirely ignored the importance of a tear in the dura adjacent to the fracture of the posterior wall of the frontal sinus. This complication requires prompt operation to prevent an ingrowth of the rapidly proliferating mucous membrane; should this take place, the membrane may become infected, producing fulminating meningitis. I [51] called attention to this in 1929. This process may have occurred in their fatal case of rhinorrhea.

SURGICAL ANATOMY AND CLINICAL PHYSIOLOGY OF AUDITORY AND VESTIBULAR APPARATUS

Surgical Anatomy and Physiology of Brain in Relation to Auditory Apparatus.—Tonndorf [52] directed attention to an error whereby the name "Trolard's vein" is applied to the vein passing from the transverse sinus or from the upper knee of the sinus over the temporal lobe to the median cerebral vein. This is really the "anastomotic vein of Labbé" (1879). The real "vein of Trolard" passes over the sylvian fossa and the central sulcus upward to the vertex. Its basal portion in the sylvian fossa is the vessel that is designated as the "vena cerebri media." The "venous anastomosis" described by Trolard in 1868 is a connection between the sinuses at the base of the brain (cavernous or petrosus superior) and the superior sagittal sinus.

Bozzi [53] reviewed the literature on the internal auditory artery in relation to arteriosclerosis and cited the importance of the latter as a determining factor in presbycusis. He made a study of the wall of the internal auditory artery and a histologic examination of this artery in its free portion in 100 patients. Finding arteriosclerosis in the auditory artery in 29.7 per cent of the patients examined who were more than 50 years old, he concluded that the increased rigidity of the peripheral vessels is an associated factor which is always preeminent in presbycusis.

It is evident that the macula sacculi must have a function totally different from that of the cochlea. Lorente de Nó [54] stated that he could not conceive that the maculae and the semicircular canals may have entirely different functions or work independently, as the whole static

51. Eagleton, Wells P.: The Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases, J. Laryng. & Otol. **44**:657 (Oct.); 721 (Nov.) 1929.

52. Tonndorf, D.: The Venous Anastomoses of Trolard and Labbé, Ztschr. f. Hals-, Nasen- u. Ohrenh. **38**:106-108, 1935.

53. Bozzi, E.: Internal Auditory Artery in Relation to Arteriosclerosis, Arch. ital. di otol. **46**:100-132 (Feb.) 1934.

54. Lorente de Nó, R.: Anatomy of the Eighth Nerve: The Central Projection of the Nerve Endings of the Internal Ear, Laryngoscope **43**:1-38 (Jan.) 1933.

labyrinth must be considered more or less as a unit because its centers in the medulla are a unit. The fibers from the three semicircular canals are said to have a different course in the medulla, but this cannot be verified by the anatomic findings. All the fibers from the semicircular canals are parallel, and the fact that they constitute two groups (Lorente de Nó's groups I and II and perhaps also a part or the whole of group III) does not signify that each group corresponds to a canal. On the contrary, group I contains thin and medium fibers and group II medium and thick fibers from all the canals. The fibers from the semicircular canals travel through the whole length of the descending root, so that each canal is represented in every part of the vestibular nuclei. The same happens with the macula.

By experiments on cats and dogs after death Jampolsky [55] found that with a pressure of from 20 to 50 cm. of water india ink injected into the caudal portion of the subarachnoid space will be deposited in the labyrinth. The route was via the aquaeductus cochleae and the perineural space of the eighth cranial nerve. Sections showed deposits in the scala tympani and the scala vestibuli throughout the coils of the cochlea and in the perilymphatic spaces of both the vestibule and the semicircular canals.

Ten similar experiments were carried out on living dogs and cats in the following manner: A suboccipital puncture was made in the usual way, 10 cc. of cerebrospinal fluid having been removed in the case of the dog and 2 cc. in the case of the cat.

The sections of the inner ear demonstrated an anatomic association between the labyrinth and the subarachnoid space, which also was via the aqueduct of the cochlea and the perineural space of the eighth cranial nerve.

However, there was a difference: Whereas the ink was deposited throughout the perilymphatic spaces in the dead animal, in the living animal it was found only in the immediate neighborhood of the aqueduct and in the scala tympani of the basal coil.

The author summarized his views as follows:

1. The subarachnoid space communicates in two ways with the labyrinth—by the aqueduct of the cochlea and by the perineural spaces.

2. The endolymphatic space is in no way connected with the perilymphatic space.

The Reticulo-Endothelium of the Aquaeductus Cochleae in the Prevention of Infection from the Labyrinth.—Tanake [56] concluded that

55. Jampolsky, L. N.: The Morphological Connection Between the Subarachnoid Space and Labyrinth, Monatschr. f. Ohrenh. **69**:23-34 (Jan.) 1935; abstr., J. Laryng. & Otol. **50**:471 (June) 1935.

56. Tanake, Masaji: On the Function of the Aquaeductus Cochleae, Jap. Ztschr. f. Oto-Rhino-Laryng., 1930, vol. 36.

the soft parts of the aquaeductus cochleae belong to the reticuloendothelial system, the cells of the aqueduct being of the fixed variety.

The aquaeductus cochleae functions to prevent labyrinthitis from within. This function is chiefly due to the reticular tissue.

The blocking of the tube caused by organization is not a direct mechanism to prevent the spread of the disease, as was hitherto generally believed, but is a secondary reaction that occurs with the proliferation of the reticular tissue by wandering histiocytes inside the tube.

Dilation of the Pial Arteries of the Parietal Lobe by Stimulation of the Geniculate Ganglion.—Investigating the question of whether higher mammals have true vasodilator nerves in the cerebral arteries, Forbes [57] and his co-workers experimented on cats. It seems probable that cats and other higher mammals possess true vasodilator innervation of the arteries supplying the parietal lobe. The nerve fibers involved come from the medulla oblongata and reach their destination by way of the facial and the great superficial petrosal nerve.

The authors' technic consisted in cutting the seventh and eighth cranial nerves immediately inside the meatus and then stimulating the facial trunk distal to the cut (close to the geniculate ganglion) with a unipolar electrode. This procedure caused dilatation of the pial arteries in the parietal lobe. That the dilatation was not due to hemorrhage and fall in blood pressure is evidenced by the one-sidedness of the lesion and by the promptness with which the reaction could be inhibited by cocaine.

Courville [58] summarized his paper on the nervus acusticus as follows: While the structure of the nervus acusticus varies considerably in the various vertebrates, the general position and relationships are remarkably constant. The peculiar structure of the nerve and the variability of related tissues in the recess account for the wide variety of neoplasms which come to occupy the cerebellopontile angle. The soft texture of its central portion and the crossing of its constituent fibers account for its frequent damage in craniocerebral injuries and susceptibility to injury and distortion by local new growths.

ANATOMY OF BRAIN AND MENINGES

Surgical Anatomy of Brain.—Functionally, the pia-arachnoid provides for the carriage of the large vessels over the surface in such a manner that only relatively small vessels actually penetrate the tissues

57. Forbes, Henry S.; Nason, Gladys I.; Cobb, Stanley, and Wortman, Ruth: Cerebral Circulation: Vasodilatation in the Pia Following Stimulation of the Geniculate Ganglion, Arch. Neurol. & Psychiat. **37**:776-781 (April) 1937.

58. Courville, Cyril B.: The Nervus Acusticus: Applied Anatomy of the Eighth Nerve and Its Environs, the Cerebellopontile Angle, Laryngoscope **42**: 415-431 (June) 1932.

of the brain. Leary and Edwards [59] pointed out that the covering of the pia-arachnoid is a relatively impermeable membrane. Blood introduced into the subdural space is confined to it and cannot escape into the subarachnoid space, though macrophages may carry pigment into the arachnoid.

Thus, the arachnoid can (*a*) limit the extension of infection from the subarachnoid to the subdural space, (*b*) retain fluid in edema and (*c*) prevent invasion by meningioma.

The subdural space does not correspond to the other serous cavities. The work of Harvey and Burr [60] showed that the pia-arachnoid fails to develop in the absence of neural crest cells, which are ectodermal. These cells apparently control the formation of the pia-arachnoid, while the mesenchyme invades and vascularizes it.

The dura, with its naked fibroblastic cells, is analogous to any exposed connective tissue surface in being prepared to form granulations and adhesions unless restrained.

Leary and Edwards [59] presented evidence to prove that:

1. The subdural space does not correspond to the serous spaces.

2. The dura is lined not by mesothelium but by fibroblastic tissue.

3. The arachnoid is covered by cells which do not appear to be mesothelial, which are probably of ectodermal origin and to which the arachnoid apparently owes its impermeability.

Embryology of the Meninges.—During its early development the nervous system is surrounded by a thin enveloping neural crest, outside of which lies the loose reticulum of the mesenchyme. The latter in most regions is derived from the mesoderm, but the experimental work of Stone [61] has definitely demonstrated the presence of ectodermal elements in the mesenchyme of the head region. Harvey, Burr and Van Camphenout [62] expressed the belief that in its later development the ectodermal primordium of the leptomeninx is in all probability added to by derivatives of the mesoderm. Therefore, it becomes a compound, mesectodermal membrane, the reactions of which to trauma and pathologic changes are determined by the particular cellular component involved. These reactions may sometimes be typically ectodermal and sometimes mesodermal and in all probability are a combination of the two.

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59. Leary, T., and Edwards, Edward A.: The Subdural Space and Its Linings, Arch. Neurol. & Psychiat. **29**:691-701 (April) 1933.

60. Harvey, S. C., and Burr, H. S.: Development of the Meninges, Arch. Neurol. & Psychiat. **15**:545-567 (May) 1926.

61. Stone, L. S.: Experiments Showing the Rôle of the Migraine Neural Crest, Arch. f. Entwcklngsmechn. d. Organ. **118**:40-77, 1929.

62. Harvey, Samuel C.; Burr, Harold S., and Van Campenhout, Ernest: Development of the Meninges, Arch. Neurol. & Psychiat. **29**:683-690 (April) 1933.

PHYSIOLOGY OF VESTIBULAR TRACT

Galvanic Falling Testing.—Although known for over a century, the galvanic falling tests have been little used for the examination of labyrinthine functions. Hitzig,[63] in 1874, noticed that the direction of the falling obtained on closing of the galvanic circuit was uniformly toward the anodal electrode. Fifteen years later, Breuer[64] localized the reactions by constructing a cerebellar as well as a labyrinthine circuit. Only with the latter circuit did the typical reactions occur. In general, galvanic nystagmus has been studied more frequently than galvanic falling, but galvanic nystagmus is inconstant and is often so small as to require a convex lens for its observation.

Galvanic Falling Reactions on a Balance Board for Testing Otolithic Functions.—To determine the integrity of the vestibulopostural arc Blonder and Davis[65] suggested the use of the galvanic falling test with the patient standing on a balance board with the eyes closed. Tested on 154 patients, it proved as accurate as the caloric tests. It was simple in execution and consistent in results.

With a group of 19 patients with supratentorial tumor Blonder and Davis found that, on an average, less galvanic current was required to produce falling than was required for patients with infratentorial tumor. They constructed the galvanic falling response as a test for otolithic labyrinthine function and expressed the hope that its wider use will lead to the diagnosis of isolated lesions in the vestibulopostural pathway.

Cortical Centers Governing Movements of the Eyeballs.—The center for stimulation of the individual muscles of the eyeball lies in the nucleus of the cranial nerve concerned, but the center for ocular movement as a functional entity lies in the cortex of the brain. Three cortical areas seem to serve as centers for conjugate deviation of the eyes. These are (*a*) the frontal lobe, where impulses for voluntary ocular movements appear to arise; (*b*) the temporal lobe, which is the center for reflex movements responding to acoustic stimuli, and (*c*) the occipital lobe, especially the angular gyrus, which serves as a relay station for reflex movements of the eyeball responsive to optic impulses.

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63. Hitzig, E.: Ueber die beim Galvanisieren des Kopfes entstehenden Stöhrungen der Muskelinnervation, in Untersuchungen über das Gehirn, Abhandlungen physiologischen und pathologischen Inhalts, ed. 1, Berlin, A. Hirschwald, 1874, p. 196.

64. Breuer, J.: Neue Versuche an den Ohrbogengangen, Arch. f. d. ges. Physiol. **44**:135, 1889.

65. Blonder, Edwin J., and Davis, Loyal: The Galvanic Falling Reaction in Patients with Verified Intracranial Neoplasms, J. A. M. A. **107**:411-412 (Aug. 8) 1936. Blonder, Edwin J.: Galvanic Falling in Clinical Use, Arch. Neurol. & Psychiat. **37**:137-141 (Jan.) 1937.

To determine how the cortical fibers entered the posterior longitudinal bundle, Spiegel [66] performed a series of experiments on animals and concluded that impulses from the frontal lobe are transmitted to this fasciculus by way of the vestibular nuclei. Thus, these nuclei serve two sets of fibers, those from the labyrinth and those from the frontal lobe, the latter conveying impulses which control the voluntary horizontal movements of the eyes. The origin of the rhythm observed in nystagmus appears, therefore, to lie in the central segment of the reflex arc which connects the labyrinth, the vestibular nuclei and the nuclei of the ocular muscles. Since neither the substantia reticularis nor the nuclei of the ocular muscles are necessary for the genesis of this rhythm, Spiegel expressed an inclination to assign to the vestibular nuclei themselves the function of originating the nystagmus rhythm. The importance of the cortically originated impulses to the vestibular nuclei is demonstrated by the fact that voluntary (i. e., cortical) movement of the eyes in the direction of the fast component of the nystagmus will increase the nystagmus.

Rôle of Vestibular Nuclei in Cortical Innervation of Ocular Muscles.—While it is generally agreed that the posterior longitudinal bundle in the pons and crus joining the corpora quadrigemina and the nuclei of the fourth and sixth nerves with the structures below carries both labyrinthine and voluntary impulses to the ocular muscles, the way in which cortical impulses enter this fasciculus is not known. As Spiegel [67] pointed out, one must assume the existence of a relay station for cortico-ocular fibers before they enter the posterior longitudinal bundle. The fact that injuries of the internal capsule or peduncle are not followed by degeneration of this bundle tends to confirm this assumption. The fasciculus contains both ascending fibers and descending fibers. The descending fibers start in the nucleus of Darkshevich (in the aqueduct), in the interstitial nucleus of Cajal and in the intracommissural nucleus of Marburg; the ascending fibers, in the vestibular nuclei. From his experimental work, Spiegel concluded that the cortical impulses for associated lateral movements of the eyes use the neurons of both vestibular nuclei to enter the posterior longitudinal bundle. This does not imply, however, that these nuclei serve for cortical innervation of conjugate vertical movements of the eyes. As long as the vestibular nuclei are intact, cortical stimulation will produce horizontal, rotary and vertical movements of the eyeballs. The neurons in the vestibular nuclei thus seem to carry impulses into the posterior longitudinal bundle from (*a*) the labyrinth, (*b*) the frontal lobe and (*c*) the occipital lobe.

66. Spiegel, E. A.: Physiopathology of the Innervation of Ocular Movements, Arch. Ophth. **8**:738-753 (Nov.) 1932.

67. Spiegel, E. A.: Rôle of the Vestibular Nuclei in the Cortical Innervation of the Eye Muscles, Arch. Neurol. & Psychiat. **29**:1084-1097 (May) 1933.

Riley,[68] reviewing the control of the ocular muscles by the central nervous system, brought to mind Bramwell's [69] contention in regard to upward movements of the eyeballs that patterns of movement and not centers for muscles are represented in the cerebral cortex.

Diagnosis of Vestibular Physiology from Cerebellar Symptoms.—The basic difference between vestibular and cerebellar signs is that the vestibular findings depend on disturbed tonus while cerebellar symptoms include asynergia and hypo-esthesia.

To distinguish vestibular from cerebellar symptoms Barré [70] pointed out that in the vestibular disturbances the spontaneous nystagmus is in the direction opposite to the deviation of the arms and trunk, whereas in the presence of a cerebellar lesion the nystagmus is toward the same side as the deviation of the arm. If the arms deviate to one side, the body deviates to the other. Cerebellar disturbance is the commonest cause of vestibular disharmony.

In the diagnosis of the vestibulospinal syndrome, Subirana [71] stressed the fact that a patient in the dorsal decubitus cannot pass unaided into another position. He expressed the belief that this symptom has not previously been described. Other evidences of this syndrome include (*a*) the accentuation of the *"marche à petits pas,"* (*b*) the fact that movements do not show marked incoordination or dysmetria, (*c*) the constancy of the deficit in the action of the psoas muscle and (*d*) the ease with which the patient lying in bed can move the lower extremities.

Barré [72] investigated the vestibular reactions during the action of scopolamine and in cases of epidemic encephalitis. No precise or clinically utilizable data were secured as a result of these examinations in cases of parkinsonism.

A case of arachnoid cyst revealed by cochlear and vestibular changes was reported by Monier-Vinard, Ramadier and Chaussé.[73]

For six years a 40 year old man had been suffering from vertigo. He was deaf in both ears, the impairment being worse on the left side. Later he com-

68. Riley, Henry Alsop: The Central Nervous System Control of the Ocular Movements and the Disturbances of This Mechanism, Arch. Ophth. **4**:640-661 (Nov.); 885-910 (Dec.) 1930.

69. Bramwell, E.: The Upward Movements of the Eyes, Brain **51**:1-17 (March) 1928.

70. Barré, J. A.: The Vestibular Syndrome and the Cerebellar Syndrome, Rev. d'oto-neuro-opht. **10**:1-11 (Jan.) 1932.

71. Subirana, A.: The Vestibulospinal Syndrome, Encéphale **26**:615-631 (Sept.) 1931.

72. Barré, J. A.: Late Vestibular Sequelae of Epidemic Encephalitis, Rev. d'oto-neuro-opht. **10**:385-395 (June) 1932.

73. Monier-Vinard; Ramadier, J., and Chaussé: Cystic Arachnoiditis of the Cerebello-Pontine Angle Revealed by Bilateral Cochlear and Vestibular Disturbances, Rev. d'oto-neuro-opht. **9**:500-506 (July) 1931.

plained of a sudden attack of dizziness in which objects appeared to turn to the right. Bone conduction was lateralized to the right ear. Caloric vestibular reactions were lost on the left side.

During the subsequent three months the deafness in the right ear improved. However, a sense of heaviness developed in the left temporo-occipital region. There was no caloric reaction from the right vertical semicircular canal.[74] He had hypesthesia of the lower part of the face on the left side, slight papilledema on both sides, anesthesia of the right cornea and slight alterations of cerebellar function on the left side. Diagnosis of tumor of the left cerebellopontile angle was made. Operation revealed an arachnoid cyst at the angle. This was evacuated, and within a month hearing had markedly improved in the right ear and the corneal reflex had returned. The left ear was still deaf.

Cause of Vertical Nystagmus.—Stengel [75] reported a case of vertical nystagmus following insulin convulsions, with an autopsy of the brain. He suggested that vertical nystagmus is due to a lesion of the oral end of Deiters' nucleus. Whether only lesions on the left side can give rise to spontaneous vertical nystagmus was not determined.

Emphasis was also laid on the danger of overdosage of insulin, particularly in patients with vascular disease. Even in apparently healthy subjects insulin may cause cerebral edema; in patients with cerebral vascular disease it may have a more deleterious effect.

A 60 year old diabetic patient suddenly became confused, showing right hemiplegia, a positive Babinski sign on the right side, paresis of the left facial nerve, hypesthesia on the right side and mild finger to nose ataxia on the left side. Spontaneous vertical nystagmus was present on fixation. It was increased in intensity with upward movement of the eyes. Necropsy showed a fresh softening in the dorsal part of the pons on the left side. The caudal end of this area implicated the oral third of Deiters' nucleus, the brachium conjunctivum and the lemnisci, both medial and lateral.

Spiegel established that the labyrinth is represented in the cortex and its cortical localization is to be sought in a part of the temporal lobe.

Spiegel [76] concluded that stimulation of the labyrinth produces epileptiform attacks if the posterior parts of the gyrus ectosylvius and the gyrus suprasylvius are made hypersensitive by strychnine poisoning. A cortical representation of the labyrinth is in the dorsocaudal parts of the temporal lobe which border on the parieto-occipital region.

It is the excitation of the labyrinth by rotation which causes the eipleptiform attacks.

Supravestibular Pathways.—In 5 of 6 patients suffering from cephalo-oculogyric crises—movement of the eyes, head and body—

74. The loss of reaction from the vertical canals, which I first described as a sign of increased intracranial pressure, persisted on the right side.

75. Stengel, Erwin: Localizing Significance of Spontaneous Vertical Nystagmus, Ztschr. f. d. ges. Neurol. u. Psychiat. **153**:417-424 (Aug.) 1935.

76. Spiegel, E.: The Cortical Centers of the Labyrinth, J. Nerv. & Ment. Dis. **75**:504-512 (May) 1932.

Shapiro [77] found jerky voluntary movements of the eyeball or weakness of the muscles of accommodation and convergence during the crisis.

He witnessed five attacks; in one there was horizontal deviation of the head and eyes; in the others the deviation was oblique.

Vestibular examinations showed spontaneous vestibular abnormality in only 1 patient. Two were found to have diminished or no susceptibility to vestibular stimulation. One had perverted past pointing. Another had absence of falling reaction and perverted vertigo. In 2 patients there was impairment of downward nystagmus induced by rotation with the head over the shoulder.

The ability to induce vestibular nystagmus during a crisis appeared proportional to the strength of the stimulus and the severity of the spasm.

Autopsy studies suggest the midbrain as the seat of pathologic changes responsible for the crisis. Shapiro's data tend to confirm Muskens' view that oculogyric crises are forced movements due to interruption of the pathways connecting the end-stations of the secondary vestibular neurons to the globus pallidus of the lenticular nucleus.

PHYSIOLOGY OF CEREBROSPINAL FLUID

Absence of Influence on Intracranial Pressure of Restriction of Fluids.—Fremont-Smith and Merritt [78] stated (*a*) that the cerebrospinal fluid pressure was not appreciably influenced by the drinking of large amounts of water when diuresis was normal, (*b*) that when the excretion of urine was prevented by pitressin, a definite rise in the intracranial pressure was obtained and (*c*) that no decrease in pressure was caused by restriction of fluid intake. However, restriction of fluid intake may cause diminution of the number of epileptic attacks, although the authors expressed the belief that this is brought on by variations in the cerebrospinal fluid pressure.

Circulation of Cerebrospinal Fluid.—Hassin [79] expressed the belief that the contents of the subarachnoid space are derived not from the choroid plexus but from the tissue fluids of the cerebrospinal parenchyma.

One may thus speak of circulation of the cerebrospinal fluid, but not as an active physiologic process, such as the circulation of the blood,

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77. Shapiro, S. L.: Otologic Findings in Oculogyric Crises: Contribution to the Study of Supravestibular Pathways, Arch. Neurol. & Psychiat. **34**:714-733 (Oct.) 1935.

78. Fremont-Smith, Frank, and Merritt, Houston H.: Influence of Variations in Fluid Intake on Intracranial Pressure in "Epileptics," Arch. Neurol. & Psychiat. **29**:454-466 (March) 1933.

79. Hassin, George B.: So-Called Circulation of the Cerebrospinal Fluid, J. A. M. A. **101**:821-823 (Sept. 9) 1933.

which is discharged by a central force into a system of elastic tubes. The tissue fluids seem to be in a state of equilibrium, and interference with them tends to produce either (*a*) abnormal currents, such as occur in spinal punctures, or (*b*) phenomena of stasis, which are manifest wherever an obstruction of the system of pathways for the fluid exists.

Physiologic Causes of Increased Intracranial Pressure.—In experiments on dogs and rabbits Guernsey, Weisman and Scott [80] found that the rise in arterial pressure following an elevation of intracranial pressure was of central origin. The carotid sinus always acted as a depressor zone, tending to keep the pressure low even when the centers needed blood. When the carotid sinus was denervated, increased intracranial pressure provoked a prompter and greater rise in arterial pressure, although the differences before and after denervation were not great.

AUDITORY AND VESTIBULAR DISTURBANCES OF TOXIC ORIGIN

In rabbits Seiferth [81] was able to elicit nystagmus by means of postural changes made during acute quinine poisoning. The quinine did not affect normal rotary and caloric irritability, and as the peripheral labyrinth was found to be intact, it is probable that the site of origin of the nystagmus must have been in the central nervous system.

Seiferth expressed the belief that the drug causes lesions in the endothelial lining of the smaller vessels and that the mechanism of postural nystagmus is thus basically vascular. Seiferth found no histologic changes in the petrous bones after the subcutaneous administration of toxic doses of quinine to dogs.

TRAUMATISM TO VESTIBULAR AND AUDITORY APPARATUS

Aural and Nasal Symptoms Associated with Injury to Restiform Body.—The syndrome of injury to the restiform body—the inferior cerebellar peduncle—first described by Mann [82] consists of (*a*) impairment of conjugate movements of the eyes, (*b*) a tendency to fall, (*c*) impairment of the associated movements of swinging and walking, (*d*) deadening of the corneal and "nasal mucosa" reflexes, (*e*)

80. Guernsey, C. M.; Weisman, S. A., and Scott, F. H.: Effect on the Reflexes of the Carotid Sinus of Raising the Intracranial Pressure, Arch. Int. Med. **52**:305-316 (Aug.) 1933.

81. Seiferth, L. B.: The Effect of Quinine on the Auditory Organ and the Vestibular Apparatus, with a Contribution to Our Knowledge of Experimental Postural Nystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:367-388, 1935.

82. Mann, L.: Injury to the Restiform Body, Deutsche med. Wchnschr. **57**: 2172-2175 (Dec. 25) 1931.

increased vasomotor irritability, (*f*) congestion in the head and (*g*) increased spinal fluid pressure. Several cases were reported by Mendel.[83] The impairment of the ocular movements and diminution in hearing tend to persist.

One of Mendel's patients was a man of 47 who was rendered unconscious by a fall on the occiput. He subsequently complained of poor hearing and tinnitus in the left ear, vomiting and convulsions. The left corneal reflex was diminished, and movements of the eyeball to the left were impaired. The galvanic nystagmus was more pronounced to the left. There was hypesthesia of the entire left side of the body. The Oppenheim sign was present in the left foot.

Mortality from Fracture of Skull with Hemorrhage or Loss of Cerebrospinal Fluid from Ear.—Gurdjian[84] found roentgen evidence of fracture of the skull in 93 per cent of the cases of injury to the head with unilateral bleeding from the ear. Of 2,600 cases of head injury, he reported 476 in which there was a discharge of blood or spinal fluid from the ear. Mastoiditis was found in 3 of these cases; meningitis developed in 4 and otitis media in 9. The death rate was 67.3 per cent among patients who had a bilateral aural discharge.

Miller and Lauppe[85] found 22 reported cases of mastoiditis following otitis media due to aural bleeding associated with head injury.

Origin of Deafness and Degeneration of Inner Ear After Trauma to Skull.—Voss'[86] patient was completely and bilaterally deaf following an injury to the skull and died from meningitis a month after the trauma. Autopsy disclosed a transverse fracture in the left temporal bone extending the entire width of the pyramid in the vestibular region. A small three-cornered sequestrum was seen near the anterior edge of the pyramid. The nerve endings in the vestibule and the semicircular canals showed atrophic degeneration without any admixture of blood. The nerves of hearing in the internal porus acusticus were torn. As a result of endolymphatic hemorrhage, secondary atrophy of the organ of Corti had occurred.

Hearing of Patients with Cerebral Injury.—In cases of head injury Gottlieb[87] found that (*a*) the hearing is reduced throughout the tone

83. Mendel, Kurt: The Traumatic Syndrome of the Restiform Body, Deutsche med. Wchnschr. **59**:529-530 (April 7) 1933.

84. Gurdjian, E. S.: Ear Complications in Acute Craniocerebral Injuries, Radiology **18**:74-82 (Jan.) 1932.

85. Miller, R. K., and Lauppe, F. A.: Mastoiditis Following Skull Fracture, J. Michigan M. Soc. **29**:912-914 (Dec.) 1930.

86. Voss, O.: Histologic Contribution to the Question of the Origin of Deafness and Inner Ear Degeneration Following Skull Trauma, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:483-490, 1934.

87. Gottleib, Mark J.: Effect of Head Injury on Hearing and Orientation, Am. J. Surg. **32**:455-462 (June) 1936.

scale or, more commonly, (*b*) the perception is less acute for high tones than for those at the middle and lower ends of the scale.

Disturbances of function of the end-organs are not as common as injuries to the pathways that lead to the brain stem and the cerebellum.

The effects of injury to the brain stem and the cerebellum are permanent and are likely to become worse with time.

I suggest that the explanation of the "continued effects of injury to the brain stem and cerebellum" may be furnished by Courville,[88] who found diffuse hemorrhagic softening of the cortex resulting from rupture of small cortical veins which was incidental to a sudden reversal of the current in these vessels or to persistent and increasing obstruction by continued pressure, which often comes on days after the original injury.

Injury to Internal Auditory Meatus.—Many microscopic fractures occur in the cribriform plates themselves, most particularly in the vestibular cribriform plate. This may be due to the fact that the temporal bone is weakest in this region because the internal and external auditory meati are here on the same line; furthermore, the bone of the cribriform plates is brittle. Fowler[89] described it as calcified connective tissue rather than true bone. It is conceivable that the same forces which produce these microscopic fractures may stretch some of the fibers of the eighth cranial nerve over the sharp edges of the small holes in the lamina cribrosa or the transverse crest and so break their primitive sheath or their endoneurium. Incidentally, the protective connective tissue coat of the nerve fibers becomes much thinner just as the fibers enter the cribriform plate.

Intracranial Pneumatocele.—Reviewing 72 cases of intracranial pneumatocele, Worms, Didiée and Grumbach[90] found that the air was derived usually from cerebral trauma that opened an accessory sinus and more rarely from the mastoid process.

The entrance of air is caused by the air pressure in the sinus or by the aspiration of the brain, the escape of air being prevented by valvular closure. Extracerebral and ventricular pneumatoceles appear soon after the injury. On the other hand, the intracerebral pneumatoceles and those which are secondary to rupture into the ventricles do not occur until weeks or months after the injury.

The source of the air could not be determined in 6 cases.

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88. Courville, Cyril B.: Diffuse Cortical Contusion of the Occipital Lobe, Arch. Path. **20**:523-534 (Oct.) 1935.

89. Fowler, E. P.: Acoustic Tumors Within the Internal Auditory Meatus, Laryngoscope **46**:616-627 (Aug.) 1936.

90. Worms, G.; Didiée, L., and Grumbach, L.: Intracranial Pneumatoceles, Ann. d'oto-laryng., May 1932, pp. 481-533.

Cured Meningitis with Pneumatocele.—Skoog [91] reported a case of cured meningitis associated with an intracranial pneumatocele.

A young man was rendered unconscious by a fall. Roentgenographic examination disclosed two curved fractures, in the left frontal bone, one of which passed across the ethmoid bone and the other across the sagittal suture. Attacks of vomiting returned a month later, accompanied by headache and general apathy. Clear watery fluid began to drip from the left nostril. When he was put to bed, he complained of more headache, but the discharge of fluid stopped. Ten days later the temperature rose. There was rigidity of the neck. Spinal fluid pressure was increased, and a high cell count was noted in the specimens of spinal fluid. Bacteriologically, a short rod-shaped bacillus was found in pure culture. Roentgenography revealed air in the cranium along the falx beside the frontal bone. The cell count gradually diminished, and the intracranial air pocket became smaller. Complete recovery occurred.

ADVANCES IN UNDERSTANDING OF CEREBRAL AUDITION

Cortical Area of Hearing.—Escardó and Horn [92] recorded the case of a 4 year old child who became deaf and dumb after an infection. On macroscopic examination the brain showed a small supratemporal surface. Microscopically, the cortex of area *TB* (von Economo's classification) was poor in cells. The vestibular and cochlear nerves were normal on the left side. The right cochlear nerve was entirely atrophied; however, the bulbar fibers were normal. Both cochlear fibers ended in the acoustic tubercle (in the lateral recess of the fourth ventricle) and in the cochlear nucleus. In the literature, changes in the auditory tract in the deaf and dumb were reported in only about one eighth of the cases.

Cerebral Auditory Mechanisms.—To determine what parts of the cerebral cortex are used in the formation and retention of auditory habits, Wiley [93] subjected rats to auditory stimuli. The rats were first trained to respond to an auditory stimulus and then were subjected to cortical operation, small destructive lesions being produced in various areas of the cortex. The rats were then retrained until they had reached the original level of learning. When this had taken place, they were killed and the brains were carefully studied. The results showed that Fortuyn's area *p*, which includes the posterolateral part of the cerebral cortex, delimits the auditory mechanism fairly closely and that extensive destruction of area *p* may abolish an auditory habit, but the habit may be reacquired.

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91. Skoog, Torsten: Intracranial Air Collection After Damage of the Pneumatic Air Spaces of the Skull, Acta chir. Scandinav. **68**:310-324, 1931.

92. Escardó, E., and Horn, L.: Further Investigation on the Cortical Architecture in the Deaf and Dumb, Ztschr. f. d. ges. Neurol. u. Psychiat. **135**:555-564, 1931.

93. Wiley, L. E.: The Function of the Brain in Audition, J. Comp. Neurol. **54**:109-141 (Feb.) 1932.

Hereditary Atrophy of Acoustic Nerves.—Hereditary atrophy of the acoustic nerves, Popow [94] suggested, is usually clinically described as congenital deafness, congenital deaf-mutism, heredodegenerative deafness, progressive labyrinthine deafness, heredodegenerative baryecoia or labyrinthine deafness of young persons. All may be grouped under the term heredopathia acoustica. On the basis of present knowledge, Popow expressed the belief that congenital deafness may be transmitted through recessive as well as through dominant patterns.

Mesencephalic Deafness Associated with Tumor of the Brain.—An interruption between the inferior colliculus of the corpora quadrigemina and the medial geniculate body (acoustic isthmus) produces so-called mesencephalic deafness. Mesencephalic deafness is usually a late symptom of pinealoma. It is commonly accompanied by hyperirritability of the labyrinth. In these cases secondary cochlear atrophy plays a rôle similar to that which it plays in deafness caused by tumor of the eighth nerve. Brunner [95] summarized the causes of mesencephalic deafness as: (1) a tumor of the mesencephalic tegmentum of the crus which develops caudally from the red nucleus (the tegmental nucleus), (2) any thalamomesencephalic tumor, (3) a pineal tumor or (4) a suprasellar tumor. A pinealoma penetrating into the third ventricle will impair auditory conduction sooner than a tumor of the quadrigeminal plate which penetrates the pulvinar.

Disturbances Associated with Abscess of the Temporal Lobe.—In 5 patients who had recovered from otogenic abscess of the temporal lobe Kompaneets [96] examined the contralateral (healthy) ear. While bone conduction for the low tones was reduced, hearing for conversation remained unchanged. Audiometry indicated a drop in the hearing curve for low tones. It thus appears that in unilateral disease of the cortical centers or the subcortical paths a decrease in perception of low tones is found in the healthy ear, which indicates that it is chiefly the fibers concerned in the perception of low tones that are involved in the crossing of the pathways of hearing. Bone conduction was also found reduced on the side opposite the abscess. This cannot be explained by the assumption that perception of sound through bone occurs only by way of the peripheral termini of the auditory nerve; it is necessary to suppose that this type of perception is, in part at least, mediated directly through the cerebral cortex.

94. Popow, N. A.: Hereditary Atrophy of the Acoustic Nerve, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:407-413, 1935.

95. Brunner, Hans: Clinical Aspects of Mesencephalic Deafness, Monatschr. f. Ohrenh. **69**:1049-1061 (Sept.) 1935.

96. Kompaneets, S. M.: Central Disturbances of Hearing in Abscess of the Temporal Lobe, Monatschr. f. Ohrenh. **67**:1322-1329 (Nov.) 1933.

SIGNS OF INTRACRANIAL HEMORRHAGE AND THROMBOSIS

Two cases of cerebral apoplexy with recovery following surgical intervention were recorded by Kaplan,[97] one of which occurred in a patient suffering from chronic aural suppuration.

A woman aged 57 had had an intermittent purulent discharge from the right ear for many years. One morning she awakened with a "bursting" sensation in the right side of the head. She vomited, felt dizzy and complained of violent headache. Examination disclosed a slight horizontal nystagmus; the blood pressure was 145 systolic and 100 diastolic; she had a tendency to turn the head to the right. Four days later she suddenly acquired left hemiplegia, stupor and stiffness of the neck. The spinal fluid was normal. In spite of the stiffness of the neck, the Kernig sign was not present. The right pupil was larger than the left. A trephine opening was made over the left postparietal region; clear fluid was obtained from the left ventricle and air was introduced. Roentgen films disclosed absence of air in the neighborhood of the right ventricle, whereas the left ventricle was moderately dilated and displaced to the left. A trephine opening was then made on the right side, and 60 cc. of old, dark blood with degenerated brain tissue was aspirated. She improved rapidly and was discharged from the hospital a month after the operation, with only slight residual paresis. This eventually disappeared, leaving left homonymous hemianopia.

Falling in Disease of Superior Cerebellar Artery.—Analyzing a series of cases of disease of the superior cerebellar artery, Critchley and Schuster [98] found the coexistence of ataxia and sensory disturbances as the most characteristic set of symptoms. The ataxia was on the side of the lesion, while the sensory phenomena were contralateral. Nystagmus was not found, but all the patients had astasia-abasia and usually fell toward the affected side.

Dysphagia in Partial Thrombosis.—Williams'[99] patient complained only of difficulty in swallowing for a week. A collection of saliva in the glosso-epiglottic fossa was visualized by laryngoscopy. He had ptosis of the left eye, slight narrowing of the left papebral fissure and slight facial weakness on that side.

Sensory examination disclosed hemianalgesia implicating the face, neck, trunk and extremities on the right side. The knee jerks were greater on the right side than on the left. The plantar reflex, normal on the left, was indefinite on the right. There was no incoordination. The Wassermann reaction was negative.

Dysphagia is a common and often persistent finding in cases of thrombosis of the inferior cerebellar artery.

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97. Kaplan, Abraham: Cerebral Apoplexy: Two Cases with Recovery Following Surgical Intervention, Arch. Neurol. & Psychiat. **37**:701-704 (March) 1937.

98. Critchley, M., and Schuster, P.: The Syndrome of the Superior Cerebellar Artery, Ztschr. f. d. ges. Neurol. u. Psychiat. **144**:681-741, 1933.

99. Williams, T. J.: Partial Thrombosis Affecting Supply of Left Inferior Cerebellar Artery: Dysphagia, J. Laryng. & Otol. **45**:883-884 (Dec.) 1930.

SUPPURATIVE INTRACRANIAL COMPLICATIONS OF OTITIC AND NASAL ORIGIN

In a study of 8,000 cases of suppuration of the middle ear Schillinger [100] found intracranial complications of spontaneous origin in 2.2 per cent.

Goldman and Shwartzman [101] reported on 168 cases of bacteremia caused by Str. haemolyticus. In 91 per cent of the cases the primary foci were located in the respiratory tract (pulmonary infection, infection of the upper respiratory tract and acute otitis media with meningitis); the infection occurred in the winter or spring months. All the cases in which the origin was otitic occurred in the winter or spring.

There were 4 patients with otitis media with meningitis who had streptococci in the blood stream. All 4 succumbed. In 3 of the 4 cases there were no secondary foci, probably because of the fulminating course of the illness. The authors consider that the meningitis was the source of the invasion of the blood stream.[102]

Deaths from Intracranial Suppurative Disease

1. Meningitis	110	
2. Meningitis and abscess of the brain	53	
3. Abscess of the brain	14	
Total	177,	or 48.8%

Pathways of Suppurative Meningitis

1. Auditory apparatus	90	
2. Paranasal sinuses	18	
3. Cavernous sinuses	8	
4. Postoperative	8	
5. Miscellaneous	3	
Total	124,	or 70%

Pathways of Abscess of the Brain

1. Auditory apparatus	37	
2. Paranasal sinuses	18	
3. Osteomyelitis, etc.	3	
4. Miscellaneous	9	
Total	67,	or 30%

100. Schillinger, Raphael: Otitic Meningitis, Arch. Otolaryng. **25**:455-464 (April) 1937.

101. Goldman, Joseph L., and Shwartzman, Gregory: Streptococcus Hemolyticus Bacteremia with Special Reference to Otolaryngologic Conditions, Ann. Otol., Rhin. & Laryng. **44**:961-973 (Dec.) 1935.

102. Eagleton, Wells P.: Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels, Arch. Otolaryng. **15**: 885-905 (June) 1932.

Statistics Concerning Intracranial Complication from Ear and Nose.—Reviewing the data concerning intracranial suppurative disease for eight years (1926 to 1933) at the Manhattan Eye, Ear, Nose and Throat Hospital by an analysis of the deaths which occurred during that period, Eggston [103] found that there were 363 fatalities. There were 110 cases of suppurative meningitis, 53 of abscess of the brain and 14 of both abscess of the brain and meningitis. Thus, there were 177 cases of suppurative intracranial disease, 48.8 per cent of all the deaths in the institution being from this cause. The majority of the patients were between the ages of 1 and 10 years.

Ten patients with abscess of the brain recovered.

There were 31 with intracranial complications following nasal sinusitis, 18 of whom died with meningitis and 8 with abscess of the frontal lobe. Five patients with abscess recovered. Of the 18 cases of meningitis, 6 were postoperative.

Frequency of Dural and Intracranial Complications of Otitis Media.—Reviewing the records of 10,000 consecutive autopsies, Courville and Nielsen [104] found 128 cases of septic meningitis. There were 2 cases of chronically thickened dura, 19 of extradural abscess, 17 of subdural abscess and 6 of pachymeningitis. There were 7 dural fistulas. In the year 1929 and 1930 complications of otitis were found in 7.3 per cent of 859 autopsies; in the year 1932 and 1933 they were present in 7.8 per cent. The portion of the complications which were intracranial was 49 per cent in the first series (1929 and 1930) and 39 per cent in the later series. Jacksonian seizures, usually contralateral and occasionally followed by weakness or paralysis, were a frequent indication of extension of the infection to the cerebrum, especially in children. In older persons this syndrome was associated more often with thrombosis of the lateral sinus. The authors expressed the belief that when this convulsive episode precedes the meningitic symptoms it suggests that the meningeal focus began in this area. When the syndrome follows thrombosis of the lateral sinus, it is probable that a retrograde infection has extended along the inferior communicating vein to the central region.

Frequency of Aural Intracranial Complications.—Scarlet fever is infrequently a cause of aural intracranial complications. Among 812 persons with scarlet fever, Seiferth [105] found 147 with disease of the

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103. Eggston, Andrew A.: Pathways of Infection in Suppurative Meningitis, Ann. Otol., Rhin. & Laryng. **43**:672-688 (Sept.) 1934.

104. Courville, Cyril B., and Nielsen, J. M.: Pathogenesis of Otogenous Intracranial Lesions, Arch. Path. **20**:952-953 (Dec.) 1935.

105. Seiferth, L. B.: A Study of Scarlatinous Otitis and Labyrinthitis and Their Intracranial Sequelae, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **29**:210-261 (Nov.) 1931.

ear; only 6 of these had endocranial complications, and of these but 1 died.

Seiferth mentioned an "initial otitis" due to a toxic capillary lesion.

Mortality from Mastoiditis with Cerebral Complications.—Reviewing 2,100 cases of acute mastoiditis in patients admitted to the Bellevue Hospital over two decades, Kafka [106] found that 126 of the patients (5.1 per cent) showed cerebral complications. Three fourths of those who had these complications died, and one fourth recovered. During the same period 1,125 patients were admitted because of chronic mastoiditis. Of these patients, 83 had cerebral complications (an incidence of 7.3 per cent). Two thirds of the patients suffering from complicated chronic disease of the mastoid died, and one third recovered.

Meningitis complicating acute or chronic purulent otitis media was almost invariably fatal. Of 55 cases of meningitis complicating acute otitis media, 49 terminated fatally, a mortality rate of 90 per cent. Among the patients with meningitis complicating chronic otitis media, the mortality rate was 90 per cent also (28 of 32). A similar result was found in cases of abscess of the brain of acute otitic origin, death occurring in 16 of 18 cases of abscess of the brain following chronic otitis; there were 11 fatal cases among 15 patients, a death rate of 73 per cent.

CEREBROSPINAL OTORRHEA

Kline [107] reported a case of spontaneous cerebrospinal otorrhea.

> The patient, aged 54, was slightly deaf in the right ear from erysipelas when he was an infant. All his life he had had occasional attacks of chills, headache and vomiting, which lasted twenty-four hours.
>
> A little impairment of his memory was noted when he was 54. Two months later he had a "cold in the head," with fever, headache and vomiting, without earache, which the family insisted was his usual "bilious spell." A week after the onset of this "cold" he suddenly had a sensation of warm fluid flowing out of his right ear as he bent to pick an object from the floor. This gave every evidence of being normal cerebrospinal fluid. The lumen of the right auditory canal was obscured by an exostosis. The whispered voice was heard by the affected ear.
>
> The ear became dry after four days' rest in bed. He felt well for a few hours but then became restless and had headache and a chill, with nausea. The temperature rose to 103.2 F. That night he became irrational and had stiffness of the neck, with a slightly positive Kernig sign. Otoscopy disclosed a blister in the region of the rivinian segment. When an effort was made to wipe the canal with an applicator, the blister was ruptured, and fluid was discharged, which showed the chemical nature of cerebrospinal fluid and contained a few cocci and leuko-

106. Kafka, Martyn M.: Mortality of Mastoiditis and Cerebral Complications with a Review of 3,225 Cases of Mastoiditis with Complications, Laryngoscope **45**:790-822 (Oct.) 1935.

107. Kline, Orom R.: Spontaneous Cerebrospinal Otorrhea, Arch. Otolaryng. **18**:34-39 (July) 1933.

cytes. Micrococcus flavus was cultured. Twitchings of the right side of the face appeared. Autopsy revealed a preformed pathway from the subdural space to a cyst in the middle ear.

A circular opening 2.5 mm. in diameter was seen in the dura and the superior surface of the right temporal bone between the petrosquamous suture and the arcuate eminence. The dura surrounding this foramen was smooth and free from adhesions. A probe inserted into this foramen passed through a fistulous tract into a sac attached to the posterior bony wall of the tympanum. A dense suppurative exudate was found over the cerebral vertex. Pus taken from the exudate yielded a pure culture of pneumococci. A section from the fistula showed brain tissue, in the center of which lay a canal about 0.25 mm. in diameter, which had a thin lining composed of cells too degenerative to be identified. "Possibly the fistula may have been the duct of a congenital cyst." The accessory sinuses were not explored.

Comment.—The cyst ruptured because of the increased pressure. This resembled, at first, the attacks of chills, headache and vomiting which he had had for years and which the family had considered "biliousness." These were probably meningeal hypothalamic episodes.

Spontaneous cerebrospinal otorrhea is extremely uncommon, and this appears to be only the third case on record. The first was that cited by Thomson [108] in 1899. Fourteen years later Canfield [109] reported a case in which pus and cerebrospinal fluid escaped from the patient's ear. This otorrhea followed six weeks of earache, but there was no history of trauma.

In Canfield's case autopsy disclosed plastic basal meningitis. The apex of the temporal bone was occupied by a large pneumatic cell containing pus and spinal fluid. A perforation through the wall of this cell had permitted infection to enter the subdural space. Through the same opening, spinal fluid escaped into the pneumatic structure of the temporal bone and thence to the middle ear and the external auditory canal.

In 1931 Roth [110] recorded a case in which a man had had two operations on the mastoid. Apparently the labyrinth was wounded in one of these.

Roth's patient, 53 years old, had had otorrhea from the left ear since childhood. After the first mastoidectomy the discharge became nonfetid, but attacks of vertigo persisted until they were relieved by the second operation.

A former associate of mine, John V. Bissett, observed a case of spontaneous cerebrospinal otorrhea which came on two years after the patient had been cured of bacterial meningitis with dural drainage through the ear. Keeping the patient at rest in an upright position

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108. Thomson, St. Clair: Cerebrospinal Fluid and Its Spontaneous Escape from the Nose, London, Cassell & Co., 1899.

109. Canfield, R. B.: Some Conditions Associated with Loss of Spinal Fluid, Ann. Otol., Rhin. & Laryng. **22**:604-622, 1913.

110. Roth, Emanuel: Discharge of Cerebrospinal Fluid into the External Auditory Canal, Arch. Otolaryng. **13**:439-442 (March) 1931.

for ten days caused cessation of the discharge. I [111] also cured a patient with spontaneous cerebrospinal otorrhea following an operation on the lateral sinus by transplantation of a piece of mucous membrane from the mouth.

OTITIC AND RHINOGENIC ENCEPHALITIS

Otogenous Encephalitis.—Roger, Crémieux and Bonnet [112] cited a case in which a patient had choked disk and encephalitic phenomena, including vertical paralysis of the ocular muscles with loss of upward movement in both eyeballs, about a year and a half after inflammation of the middle ear had been cured by operation. After operative intervention, which revealed no abscess of the brain, the patient recovered.

Although the authors designated this as "otogenic" encephalitis, I feel that the later symptoms could not have been due to otitic suppuration.

Nonsuppurative otogenous encephalitis is probably more frequent than is suspected. It is characterized by continuous high fever and by headache, a focus of osteitis being indispensable for its production.

Bijleveld [113] recorded a case of bilateral mastoidectomy with postoperative fever of the pyemic type, with marked local signs (mimic facial paralysis on the left side). The right temporal lobe was explored; no pus was found, but softened cerebral tissue was aspirated through the exploring needle. After a radical mastoidectomy the patient made a prompt and complete recovery.

Horgan's [114] case illustrates the difficulty of distinguishing abscess of the brain from localized serous meningitis (otitic hydrocephalus) and nonsuppurative encephalitis.

The patient, a 16 year old boy, complained of pain in the right ear with chronic suppurative otitis media and suffered from vomiting, attacks of coma and headache on the right side. The left knee jerk was exaggerated, and the left pupil was sluggish and dilated. There was no papilledema. Left hemiplegia and weakness of the left lower part of the face were present. A mastoidectomy was performed. As the hemiplegia continued, exploration of the sinus, cerebellum and temporal lobe was made, with negative results. Spinal puncture gave negative results. The condition remained unchanged for several days and then began to improve after a crucial incision of the congested dura. Thereafter there was slow and gradual improvement of the symptoms. Eight weeks after the original operation the boy was discharged with a dry ear and free from symptoms.

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111. Eagleton, W. P.: Unpublished data.

112. Roger, Henri; Crémieux, Albert, and Bonnet: Nonsuppurative Otogenic Temporal Encephalitis with Conjugate Paralysis of Ocular Muscles and Loss of Upward Movements of Both Eyeballs; Recovery, Rev. d'oto-neuro-opht. **10**:332-336 (May) 1932.

113. Bijleveld, J. A.: Nonsuppurative Otogenous Encephalitis of the Temporal Region, Rev. d'oto-neuro-opht. **14**:81-85 (Feb.) 1936; abstr., Arch. Otolaryng. **25**:102 (Jan.) 1937.

114. Horgan, J. B.: Localized Non-Suppurative Encephalitis of Otitic Origin, Brit. M. J. **2**:161-162 (July 27) 1935.

Horgan concluded that the patient had nonsuppurative encephalitis of otitic origin, which was probably an early inflammatory predecessor to an abscess of the brain, which would have occurred had it not been for the mastoidectomy, decompression and exposure of the brain.

Cataphoretic Velocity of Streptococci in Nonsuppurative Encephalitis.—Rosenow and Jensen [115] expressed the belief that streptococci sharing the same neurotropic cataphoretic velocity are, directly or indirectly, causative of encephalitis, because of their behavior on cultures of dextrose-brain broth when an electric current is turned on.

NEURALGIC OTALGIA

Disturbances of Geniculate Ganglion.—The clinical classification of Ramsay Hunt's [116] syndrome allows for four subgroups: (1) herpes oticus, (2) herpes oticus with facial palsy, (3) herpes oticus with facial palsy and impaired hearing and (4) herpes oticus with facial palsy and Ménière's syndrome. Each type depends on focal disease of the geniculate ganglion. Because of their close proximity to this ganglion the seventh and eighth cranial nerves may become involved. The outstanding feature of the syndrome is the herpetic eruption, which is variably distributed over the ear drum, external auditory canal, concha, tragus and lobule.

Geniculate ganglionitis is a rare disease. Gruber, whose work is cited by Hunt,[116] found only 5 instances among 29,000 patients with aural disease. During a ten year period in which 46,000 cases of otologic disease were observed, only 2 cases of herpes auris were recorded in the Manhattan Eye and Ear Hospital. In the New York Eye and Ear Infirmary only 6 cases were recorded in twenty years.

The disease can be identified by (1) the intense neuralgic pain with (2) the appearance of a herpetic eruption in a characteristic location.

A case of inflammation of the geniculate ganglion associated with histologic evidence of otosclerosis and bilateral labyrinthitis was presented by Maybaum and Druss.[117]

A 51 year old man had a history of diminished hearing without discharge in the left ear for a year and a half. He suddenly acquired a disturbance in the sense of smell, and two days later partial palsy of the right side of the face occurred, followed by total deafness in the right ear. A swelling in the throat associated with dysphagia appeared five weeks later. The right side of the head was swollen, and a red bulging and tender area was also noticed on the inner aspect of the

115. Rosenow, E. C., and Jensen, L. B.: Cataphoretic Velocity of Streptococci in Encephalitis, J. Infect. Dis. **52**:167-184 (March-April) 1933.

116. Hunt, J. Ramsay: A Further Contribution to Herpetic Inflammations of the Geniculate Ganglion, Am. J. M. Sc. **136**:226-241 (Aug.) 1908.

117. Maybaum, J. L., and Druss, J. G.: Geniculate Ganglionitis (Hunt's Syndrome), Arch. Otolaryng. **19**:574-581 (May) 1934.

right ankle. Examination disclosed peripheral facial paresis on the right side, with impairment of taste on the anterior two thirds of the right side of the tongue. Hearing was totally lost in the right ear, and there was partial nerve deafness in the left. Caloric tests revealed total loss of irritability of the right vestibular nerve. The whole picture suggested complete peripheral involvement of both divisions of the right eighth cranial nerve. A small amount of pus containing Staph. aureus was procured by irrigation of the right antrum.

For six weeks the condition remained static; then abruptly the temperature rose to 102 F. and pain was experienced in the left ear. A large bleb on the left ear drum partly occluded the external canal, with other blebs on the left auricle and pinna. Partial paralysis of the left side of the face developed two days later. The condition was diagnosed as Hunt's syndrome. After twelve days the blebs gradually disappeared. A month later the patient acquired epididymitis on the right side, and shortly thereafter he complained of backache. He rapidly declined and died four and one-half months after the initial disturbance in the sense of smell.

The changes observed at autopsy were periarteritis nodosa involving the kidneys, testicles, heart and mesenteric vessels; multiple periarteric aneurysms of the kidneys, some of which had ruptured into the renal pelvis; chronic purulent antritis on the right side; chronic emphysema and bronchitis, and parenchymatous degeneration of both the liver and the myocardium.

The histologic studies of the cranial nerves revealed the following picture: The cells of the left geniculate ganglion were disintegrated and enveloped by capsule cells. The left facial nerve near its ganglion showed advanced degenerative changes, while in the left eighth cranial nerve similar but less marked changes were noted. An occasional gitter cell with lymphocytic infiltration was seen in relation to the eighth cranial nerve. The meningeal sheath of this nerve was infiltrated with mononuclear elements. At one end of the eighth nerve the cells in Scarpa's ganglion were implicated in an extensive degenerative process.

The mucosa lining the left tympanic cavity was thickened and showed inflammatory changes. A dense network of fibrous tissue at the niche of the round window extended into the scala tympani. There were inflammatory changes in the mucosa lining the peritubal and periantral cells and in the pneumatic spaces of the petrous pyramid. Fresh hemorrhagic exudate lay in the first vestibule, cochlea, utricle and semicircular canals. Purulent exudate in the first whorl of the left cochlea had undergone organization and fibrosis. A patch of otosclerosis was noted at the typical site in the region of the oval window.

There was a marked suppurative process, with necrosis of the mucosal lining in the right tympanic cavity and the adjacent pneumatic spaces. An old hemorrhagic exudate was found in the internal auditory meatus and in the fallopian canal about the right geniculate ganglion. An area of otosclerosis was present in the region of the right oval window.

On neither the right nor the left side could any evidence of periarteritis nodosa be found in the cranium. A thorough search for this was made, some of the sections including the apex of the petrous pyramid.

The history of pain in the ear and fever followed by blebs in the auditory canal, on the ear drum and on the auricle, with impairment of hearing and partial facial palsy, is suggestive of Hunt's syndrome of geniculate ganglionitis. The degenerative changes in the geniculate ganglion, while not exclusively associated with this syndrome, are con-

sistent with it. In some cases of geniculate ganglionitis, the cells show nondegenerative inflammatory changes, while in other conditions, notably the so-called "rheumatic" disorders of the facial nerve, the same cells may exhibit degenerative alterations. Pathologic evidence of bilateral suppurative labyrinthitis in the absence of any clinical symptoms of labyrinthine irritation is an unusual finding. Curiously, there was loss of labyrinthine function on the right side, although the ganglionitis was on the left. The labyrinthitis probably was an extension of suppuration of the middle ear in this case.

Herpes Zoster Oticus.—Fenton [118] mentioned that by injecting material from herpes into experimental animals one is able to produce encephalitic changes but not herpetic manifestations. This accords with the observations of Olitsky [119] and his co-workers that the neurotropic action of the encephalitic virus can be converted into a dermotropic force by subdermal pad inoculations. In this manner, the virus of stomatitis can be made neurotropic and, in mice at least, will pass from the nasal mucosa to the meninges and the brain.

Sinusitis affecting the sphenoid or the posterior ethmoid sinuses may cause severe pain in the distribution of the facial sensory nerve, relayed at the geniculate ganglion from the great superficial petrosal and the vidian nerve. Somogyi [120] expressed the belief that an irritative or malignant lesion of the pharynx, larynx or basilar process may occasionally cause a similarly distributed type of pain.

OTITIC HYDROCEPHALUS

In otitic hydrocephalus there is an excessive amount of spinal fluid. The most striking feature of the condition is swelling of the optic disks. A similar degree of swelling is rarely seen in cases of abscess and is then accompanied by extreme drowsiness and malaise. Between the attacks of headache and vomiting the patient is likely to be alert, lively and of good appetite. In otitic hydrocephalus there are high pressure and normal fluid. The treatment is by lumbar puncture. Enough fluid should be removed at each puncture to bring the pressure to normal, and this should be repeated as long as there is evidence of increased pressure.[121]

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118. Fenton, Ralph: Diagnostic Factors Concerning Herpes Zoster Oticus, J. A. M. A. **103**:468-471 (Aug. 18) 1934.

119. Olitsky, P. K.; Cox, H. R., and Syverton, J. T.: Comparative Studies on Viruses, J. Exper. Med. **59**:159 (Feb.) 1934.

120. Somogyi, L.: The Otolaryngologic Picture of Herpes Zoster, Ztschr. f. Laryng., Rhin. & Otol. **23**:436-440, 1932.

121. Symonds, C. P.: Otitic Hydrocephalus: Report of Three Cases, Brit. M. J. **1**:53-54 (Jan. 9) 1932; abstr., Arch. Otolaryng. **17**:421 (March) 1933.

INTRAMEATAL FACIAL PALSY

Intrameatal Paralysis of Facial Nerve and Labyrinthitis.—Staphylococci have the ability to be reactivated after a long period of hibernation. In the case observed by Thomas and Girard [122] staphylococci from the infected humerus presumably migrated to the labyrinth capsule, where they remained quietly until activated.

A patient had several operations for osteomyelitis of the humerus over a period of eight years. Three years after the last operation vertigo, deafness in the right ear and almost total paralysis of the right facial nerve developed. A few months later she had pain in the teeth and the ear. The spinal fluid showed 700 cells per cubic millimeter. The next day the ear discharged and there was a positive Kernig sign. At operation an extradural abscess was found, which was connected to the tympanum by a fistula through the oval window. The labyrinth was opened and drained. At autopsy a fistula was found opening on the endocranial surface. The connective tissue sheath of the facial nerve was thickened and contained nuclear masses, which were composed of plasmocytes, fibroblasts and mononuclear and polymorphonuclear cells.[123]

Paralysis of Facial Nerve from Apicitis.—After having had otitis media on the left side for a month, a 9 year old girl, who was observed by Myerson and his associates,[124] suddenly acquired ipsilateral facial palsy. Mastoidectomy was done. A week later the paralysis of the facial nerve disappeared. Twenty-three days after the operation paralysis of the left sixth nerve developed, with frontal headache on the left side. At exposure of the apex an erosion was found through the roof of one of the cells. This opening was plugged by a granuloma. The headache ceased only to return a fortnight later with evidences of acute meningitis. Str. haemolyticus was cultured from the spinal fluid. The patient died the day after the onset of this infection. An abscess of the temporosphenoid lobe was found at autopsy. There had never been any sign of this abscess at any time during the patient's illness.

MÉNIÈRE'S DISEASE

Reviewing 135 cases of Ménière's disease, Mygind and Dederding [125] stated that because of the similarity in neurologic manifestations Ménière's disease is mistakenly diagnosed in many cases as tumor of the brain or encephalitis. Sometimes only the ultimate course of the disease will make it possible to eliminate the more serious intracranial disorders. They found headache in 91 per cent of the patients, usually localized to the side of the affected ear. The headache was described

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122. Thomas, André, and Girard, Louis: Facial Paralysis and Labyrinthitis: Anatomic Examination of the Facial Nerve, Rev. d'oto-neuro-opht. **11**:664-672 (Nov.) 1933.

123. I first described an intrameatal type of facial palsy as diagnostic of apical suppuration before the Southern Medical Association at Richmond, Va., on Nov. 15, 1933.

124. Myerson, Mervin C.; Rubin, H. W., and Gilbert, J. G.: Considerations on Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:62-89 (July) 1935.

125. Mygind, S. H., and Dederding, Dia: Phenomena Pertaining to the Nervous System in Ménière's Disease, Acta oto-laryng. **14**:343-355 (March) 1930.

as paroxysmal, varying with the intensity of the disorder in the ear. It was often worst in the morning. In only 7 of the patients was there any tenderness over the mastoid. Abnormalities in the cell count or the albumin content of the spinal fluid were common, and increased spinal fluid pressure was the rule. Mental symptoms, including irritability, depression and amnesia, commonly occurred during the attacks. Nearly all the patients complained of a feeling of exhaustion. Their thesis is that in Ménière's syndrome the fluid system in the labyrinth cavity communicates with and is responsive to changes in the cisterna and the ventricles and that intracellular edema, probably localized in the meninges, is transmitted to the labyrinthine fluid system, evoking parallel changes in the highly sensitive nervous elements associated with the latter. Since bulbar symptoms were rare, the authors expressed doubt as to whether the disease has its origin in the medulla oblongata.

Sagittal Headache in Ménière's Syndrome.—Among 11 patients suffering from Ménière's symptom complex, Cohen [126] found headache characteristically localized in the sagittal plane, frontal, vertical or occipital. This sagittal headache is in or over the surface projection of the falx cerebri and the tentorium cerebelli, both of which are reduplications of the dura mater. In 9 of the 11 cases there were definite neurologic signs other than those referable to the disease of the middle ear. This suggests a primary organic neurologic basis. Such a neurologic basis, however, need not necessarily explain the headache, since this symptom may be of circulatory origin. But the fact that in 7 of the cases the headache was present from the beginning of the symptoms speaks for a single origin common to the vertigo, tinnitus, deafness and headache.

Condition of Inner Ear.—Eisenberg,[127] in an effort to clarify the vestibular and cochlear function of patients with Ménière's syndrome, studied 33 cases. He concludes that Ménière's syndrome is due to a structural change in the nervous apparatus of the internal ear. It is not a definite entity but may appear in various diseases (syphilis, leukemia, infectious diseases, gout or arteriosclerosis). The cause, perhaps angioneurosis, is not definitely known.

Angiospasm as Cause of Vestibular Tract Vertigo.—The sense of numbness in the cheek and ear often accompanying attacks of vertigo is commonly known as "dead cheek." Collet [128] explained it on the basis

126. Cohen, L. D.: Headache as a Feature of Ménière's Symptom Complex, Guy's Hosp. Rep. **85**:215-224 (April) 1935; abstr., J. A. M. A. **105**:1079 (Sept. 28) 1935.

127. Eisenberg, M. D.: The Clinical Condition of the Internal Ear in Ménière's Syndrome, Monatschr. f. Ohrenh. **70**:22-36 (Jan.) 1936.

128. Collet, F. J.: Phenomenon of "Dead Cheek" Accompanying Ménière's Vertigo, Rev. d'oto-neuro-opht. **13**:665-671 (Nov.) 1935.

of angiospasm of the vestibular centers or the pathways extending into adjacent territory, especially of the fifth cranial nerve. Paralysis of the facial nerve accompanying Ménière's vertigo may be explained in the same way.

Arterial Loops in Ménière's Disease.—Dandy's [129] series of operations on the eighth cranial nerve has now reached 170, 10 being bilateral. There has been no death, and except for 4 instances of early facial palsy no after-effects have been noted in the cases of unilateral involvement. In perhaps 30 per cent of the whole series the gross lesion was also demonstrable.

Dandy said that the vascular variations in the region of the auditory nerve are such that one has difficulty in establishing the normal. Not the least surprising is that an internal auditory artery is too infrequently found and when present is of tiny caliber. A small artery does not give the impression of any casual relationship, but when the artery is more than half the size of the nerve and is directly on the nerve, it is difficult to believe that the artery is not affecting the hearing and producing the attacks of dizziness.

The actual percentage of large compressing arteries is approximately 10. It is known that probably in from 8 to 10 per cent of the total number of cases of Ménière's disease the involvement is bilateral. It is because the arteries become more rigid with age that the attacks of dizziness, deafness or tinnitus come so late in life.

Independently McKenzie has also been impressed with the significance of the arterial loops along the auditory nerve. He noted in 6 of his last 14 operations that an abnormally situated anterior inferior cerebellar artery "passed through or was lying upon the nerve."

Wittmaack found concretions in the ductus cochlearis of 2 patients who had had Ménière's disease. In 1 of these cases there was also a minute neuroma in the cochlea. Just cited a paper by Zange, who found a small hematoma in the brain stem near one of the nuclei of the auditory nerve; the vestibular canals and the cochlea showed no gross abnormality.

Dandy expressed the belief that only lesions of the sensory root of the auditory nerve (its vestibular division) can cause Ménière's disease and consequently lesions in the semicircular canals, such as concretions, would not be a cause. Although in his opinion concretions in the cochlear aqueduct cannot cause recurring attacks of dizziness and loss of hearing, he said that one cannot, categorically, deny that they may do so. That pressure in the lateral cistern does not in any way affect the semicircular canals or the organ of Corti is easily shown

129. Dandy, Walter E.: Pathologic Changes in Ménière's Disease, J. A. M. A. **108**:931-938 (March 20) 1937.

by the absence of effect from tumor of the brain, which is known always to produce a rise of pressure in the lateral cistern but which never causes any pathologic effect on hearing or on the vestibular function.

Polycythaemia Rubra Vera with Symptoms Resembling Ménière's Disease.—Polycythaemia vera, or polycythaemia rubra,[130] is a chronic, slowly progressive disease characterized by splenomegaly, cyanosis and an increase in the red cell count. The physiologic basis is a stimulation of the hematopoietic system without a parallel stimulation of the cell-destroying organs. It usually affects middle-aged men, producing a syndrome of weakness, vertigo, nausea and mental symptoms. Curiously, in the first case of polycythemia described in the literature the picture as reported by Vaquez [131] was that of vertigo, tinnitus and staggering, strongly suggesting Ménière's disease. The only satisfactory methods of treatment are irradiation over the long bones and the administration of phenylhydrazine hydrochloride.

AURAL VERTIGO

Types of Vertigo.—From examination of 300 persons suffering from head injury Grove [132] concluded that: 1. True "turning vertigo" (accompanied by nystagmus) occurs with considerable frequency in cases in which the peripheral vestibular mechanism appears to be functioning in a normal manner. This, he stated, is due to the fact that in these cases the damage to the vestibular apparatus is located beneath the floor of the fourth ventricle and not in the labyrinth. 2. The vertigo occurring in destruction of the labyrinth by a transverse fracture of the pyramid is a violent and continuous dizziness which persists for about a week and then slowly improves. 3. A third type of vertigo arises from a vasomotor disturbance in the central vestibular domain. This is neither continuous nor seriously disabling, as it occurs in transient paroxysms precipitated by exertion, ingestion of alcohol, exposure to sunlight or sudden changes in posture. 4. "Lateropulsion" is a less frequent form of tactile vertigo, described by Nageotte and Babinski.

Shapiro [133] listed the following type of vertigo arising from disturbances of the circulatory system: 1. Vasomotor instability without evidence of structural abnormality in the blood vessels is the basis in most of the so-called cases of Ménière's syndrome. Vasomotor disturbances probably are the cause of the prodromal vertigo of epilepsy

130. Sloan, Leroy H.: Polycythemia Rubra Vera, Arch. Neurol. & Psychiat. **30**:154-165 (July) 1933.

131. Vaquez, Henri: Compt. rend. Soc. de biol. **44**:384, 1892.

132. Grove, W. E.: The Symptom of Vertigo, Arch. Otolaryng. **14**:177-180 (Aug.) 1931.

133. Shapiro, S. L.: Vertigo as a Syndrome in Vascular Diseases, Illinois M. J. **70**:512-516 (Dec.) 136.

and of migraine accompanied by transient loss of half the field of vision and violent vestibular upsets. 2. Any blood dyscrasia may cause vertigo from inadequate oxygenation of the vestibular structures, such as that which occurs with leukemic deposits or with degenerative changes in the internal ear. 3. Patients with circulatory disturbances accompanied by organic changes in the blood vessels of the internal ear or the brain (including hypertension) are particularly likely to complain of dizziness on stooping or on arising quickly, because of the inability of the sclerosed vessels to adjust themselves quickly to the effects of gravity, with resultant transient anemia. 4. In association with cardiac abnormalities either a retarded or an accelerated vertigo represents a mild form of cerebral anemia due to deficient heart action.

Factors in Production of Vertigo.—McMurray [134] pointed out that vertigo is not primarily an otologic problem. Among the suggested precipitating factors are disturbed water metabolism (Dederding), sensitivity to sodium (Furstenburg, Lashmet and Lathrop), toxins from a distant focus and protein sensitization.

Ramadier and Caussé [135] stated that the absence of vertigo in the presence of intense spontaneous nystagmus is presumptive evidence of a central disturbance.

Aural Vertigo Relieved by Section of Auditory Nerve.—Four patients with aural vertigo were successfully treated by division of the auditory nerve by Cairns and Brain.[136] All had been losing hearing in one ear prior to the first attack. The attacks consisted of sudden severe vertigo, with a sense of falling in 1 case and actual falling in the other 3. External objects appeared to rotate. All the patients were practically deaf in one ear at the time of operation, the hearing in the other ear being substantially normal or but moderately affected. After the operation all 4 patients were free from attacks of vertigo and were able to resume work.

OPERATIVE TECHNIC

Repair of Injury to a Venous Sinus.—Bailey [137] reported a case of head injury with a wound of the superior longitudinal sinus. The bleeding in the sinus was controlled by pressing a piece of muscle from the patient's leg on the opening. The sinus, although lined by

134. McMurray, J. B.: Clinical Cases in Which Vertigo Is a Cardinal Symptom, Arch. Otolaryng. **22**:277-284 (Sept.) 1935.

135. Ramadier, J., and Caussé, R.: Technique of Vestibular Examinations in Neurology, Rev. d'oto-neuro-opht. **10**:305-324 (May) 1932.

136. Cairns, H., and Brain, W. R.: Treatment of Aural Vertigo by Section of the Auditory Nerve, Lancet **1**:946-952 (May 6) 1933.

137. Bailey, Percival: Wounds of the Superior Longitudinal Sinus, S. Clin. North America **9**:395-405 (April) 1929.

endothelium, is really a lacuna lying where one sheet of dura mater joins another at a right angle.

The superior lateral veins of the cerebral hemispheres unite into four principal trunks: frontal, precentral, postcentral and occipital. The postcentral trunk, which drains the central gyri, is usually the largest. Three lacunae that project over the lateral and mesial surfaces of the hemisphere receive these veins.

The most practical management of wounds of the venous sinuses is inversion of the outer wall by pushing into the sinus some rounded object which can be retained by pressure or by suture. This technic, as Bailey stated, was first recommended by Macewen and later applied by me [138] to the lateral sinus.

Technic of Cisternal Puncture.—Because of the hazard of cisternal puncture by the indirect method and because of the anastomoses connecting the internal and external venous vertebral plexuses, Astrachan [139] expressed a preference for performing this procedure by the direct, or Ayer, technic. The selection of the point of puncture is made with the patient erect, since this offers clearer landmarks. During puncture the patient lies on his right side, with the head moderately flexed and held in place by an assistant. Injury to the skull and tumor of the brain, conditions which might displace vital structures toward the median line, are contraindications to cisternal puncture.

Technic of Lumbar Puncture.—To reduce headache, nausea, vertigo and other untoward reactions associated with spinal tap, Kulchar and King [140] suggested the use of sodium amytal. Three grains (0.19 Gm.) is administered by mouth a half-hour prior to the puncture. Among patients who received this medication, the proportion of unpleasant postpuncture reactions was 13.5 per cent, whereas among ambulatory patients receiving no preliminary sedative, the proportion was 25.5 per cent. Almost half of the latter group were rigid and apprehensive during the tap, but only 2.8 per cent of the patients receiving the sodium amytal were tense or fearful.

POSTOPERATIVE ACCIDENTS

Hernia Cerebri.—Discussing cerebral hernia after operation on the ear, Hirsch [141] pointed out two major etiologic factors: (1) opening of

138. Eagleton, Wells P.: An Original Device for the Control of Hemorrhage from the Large Sinuses of the Brain by the Involution of the Outer Wall into the Lumen, M. Rec. **95**:274 (Feb. 15) 1919.

139. Astrachan, G. D.: Cistern Puncture: General Review and Comparison Between Direct and Indirect Methods, Am. J. Syph. **16**:321-334 (July) 1932.

140. Kulchar, George V., and King, Allen D.: Sodium Amytal in the Prevention of Reactions Associated with Lumbar Puncture, Arch. Neurol. & Psychiat. **30**:170-174 (July) 1933.

141. Hirsch, M.: Prolapse of the Brain After Ear Operations, Ztschr. f. Laryng. Rhin. **19**:338-340 (May) 1930.

the dura and (2) internal pressure which forces the cerebral contents outward. Prolapse of the brain will not occur, even in the presence of a fairly large dural opening, if intracranial pressure is normal. Hernia not infrequently occurs gradually during the postoperative treatment, owing to the increase in the intracranial pressure which develops as a consequence of inflammation of the brain and meninges. Enlargement of the bony defect is a method of treating prolapsus cerebri. The purpose of widening the opening is to relieve the edema due to mechanical pressure of the edge of the bony ring on the brain.

Death from Lumbar Puncture.—In a case recorded by Northington [142] a 34 year old man had been noticing intermittent drainage from the right ear for ten years. For five weeks he had attacks of vertigo and deafness and tinnitus in that ear. Caloric testing on the right side revealed a vertical upward nystagmus in the "face-up" position. Hearing on the right side was slightly impaired. At first there was no neurologic evidence of a lesion in the brain, but reexamination two days later gave the impression of an abscess of the right temporal lobe. At lumbar puncture the spinal fluid, which was under increased pressure, was found to contain 50 lymphocytes per cubic millimeter. The patient died a few hours after the spinal tap and presumably as a result of it.

Nash [143] has collected 7 cases of abscess in which death resulted from respiratory failure associated with herniation of the cerebellum into the foramen magnum which was ascribed to spinal tap intended to relieve increased intracranial pressure. Death ensued shortly after the puncture, the shortest interval being five minutes and the longest an hour and a half. In all the cases autopsy disclosed herniation of the cerebellum into the foramen magnum, with secondary compression of the medulla oblongata.

Hemorrhage into Fourth Ventricle and Death from Cisternal Puncture.—In Vonderahe and Haberman's [144] case a cisternal puncture for the diagnosis of meningitis was performed. At a second puncture no fluid was procured; the patient died four hours later.

Postmortem examination disclosed two needle puncture wounds in the medullary portion of the brain stem, with a surrounding area of hemorrhage that extended into the fourth ventricle.

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142. Northington, Page: Observations on Some Disturbances in Vestibular Function, New York State J. Med. **35**:56-65 (Jan. 15) 1935.

143. Nash, Stewart C.: Cerebellar Hernia as a Cause of Death, unpublished report presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Jan. 7, 1937.

144. Vonderahe, A. R., and Haberman, F. C.: Injury of the Medulla in Puncture of the Cisterna Magna, Arch. Neurol. & Psychiat. **29**:166-167 (Jan.) 1933.

DIAGNOSIS OF INTRACRANIAL COMPLICATIONS

Intracranial Calcification in Diagnosis of Intracranial Complications.—Loew-Beer [145] found that roentgenologically visible calcification in the dura is the result of retrogressive changes in the tissues following an inflammatory process or hemorrhage. In chronic suppuration of the middle ear dural calcification may occur above the tegmen tympani.

Influence of the Type of Mastoid Bone on Production of Cerebral Complications.—Husik [146] said that the sclerotic type of bone shows a much greater tendency to produce intracranial complications than do other forms, because compact bone more readily transmits infection to the dura.

Increased Cholesterol Content of Spinal Fluid in Meningitis and Abscess of the Brain.—Plaut and Rudy [147] considered 0.2 mg. per hundred cubic centimeters to be the upper normal limit for the cholesterol content of the cerebrospinal fluid. The quantity seemed to depend on metabolic processes in the nervous system rather than on the cholesterol content of the blood. Conditions causing an increase in the amount of cholesterol include cerebral arteriosclerosis, senile disease of the brain and some forms of neurosyphilis, notably untreated dementia paralytica. Even more marked increases are seen in meningitis, abscess of the brain and Christian-Schüller's syndrome.

Facial Spasms.—Weingrow [148] pointed out that spasm induced in facial structures by tapping is not a simple reflex of the seventh nerve. It is in reality a trigemino-facial reaction. The sensory portion of the fifth nerve comes in contact with the substantia gelatinosa of Rolando (the substance which sheathes the posterior horn of the spinal canal) in the upper two cervical metameres. The reflex can be employed to differentiate between localized and diffuse cerebral lesions. The reflexes were found of value in differentiating hemorrhagic hemiplegia from encephalitic hemiplegia. In the latter tapping one side of the face provoked a bilateral reaction, whereas in cerebral vascular disease the reaction is only ipsilateral. Weingrow found it possible to distinguish between basilar meningitis and abscess of the brain when the cranial nerves were involved.

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145. Loew-Beer, A.: Intracranical Calcification in the Roentgen Picture, Fortschr. a. d. Geb. d. Röntgenstrahlen **45**:420-449 (April) 1932.

146. Husik, David N.: Sclerotic Mastoiditis and Intracranial Complications, Laryngoscope **42**:519-530 (July) 1932.

147. Plaut, F., and Rudy, H.: The Cholesterin Content of the Cerebrospinal Fluid, Ztschr. f. d. ges. Neurol. u. Psychiat. **146**: 229-261, 1933.

148. Weingrow, Samuel M.: The Trigeminocervical Reflex, Laryngoscope **45**:375-388 (May) 1935.

(To be concluded)

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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CHRONIC PROGRESSIVE DEAFNESS INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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OTOSCLEROSIS

Of great interest in the literature on otosclerosis for 1936 is a contribution by T. H. Bast [1] on the development of the otic capsule, with special reference to fetal and infantile changes in the region of the fissula ante fenestram and their probable relation to the formation of otosclerotic foci.

Since 1930, under the auspices of the Research Council of the American Otological Society, T. H. Bast has studied the fissula ante fenestram, that tiny cleft which lies in the bone just in front of the oval window and in the region in which otosclerotic foci most often develop. It extends from the vestibule to the middle ear, is filled with connective tissue and is surrounded by a narrow rim of cartilage. The normal fissula varies from a narrow slit to a large and massive area. In some children's ears the fissula has been found to be partly or entirely filled with a cartilaginous mass, and in some adults an osteoid mass has been found to fill the fissula (Anson and Martin, Wilson). In 32 human fetal ears and 20 ears from children varying in age from 3 days to 6 years, Bast found that this peculiar cartilaginous or osteoid mass had developed in the fissula in a third of the cases. The earliest fetal age at which one of these was found was 21 weeks. At this age the otic capsule is nearly completely ossified, except for narrow rims of caritlage around the oval window and the fissula. This cartilage seems quiescent or dormant. From this inactive rim of cartilage around the fissula active cartilage arises in certain instances, apparently in an attempt to reduce the size of the fissula, since it occurs usually at its broadest

1. Bast, T. H.: Development of Otic Capsule: Fetal and Infantile Changes in Fissular Region and Their Probable Relationship to Formation of Otosclerotic Foci, Arch. Otolaryng. **23**:509 (May) 1936.

point. The active cartilage, differing histologically from the inactive rim of cartilage from which it grows, later ossifies, with the formation of atypical and unstable osteoid tissue, which Bast suspects represents the first stage in the formation of otosclerotic bone. A still later attempt to resorb and reorganize this area of atypical bone may result in a typical focus of otosclerotic bone.

Clinical otosclerosis is a disease of adult life hardly ever observed before puberty. But while clinical otosclerosis with ankylosis of the stapes to the oval window is a common condition, for every clinical case there are several specimens showing the histologic changes of otosclerosis without involvement of the footplate of the stapes when the temporal bones of adults are examined histologically in routine autopsies (Guild). The work of Bast suggests that these foci of otosclerosis, with or without ankylosis of the stapes, are the result of changes beginning in the fissula ante fenestram in fetal life in an attempt, apparently, to close the fissula.

A discussion by M. Weber [2] of the influence of heredity in otosclerosis advances a new theory, which considers otosclerosis from the point of view expressed by Bast, namely, that for every person with clinical ankylosis of the stapes there are other persons showing otosclerotic foci without clinical symptoms. Weber says that one must consider otosclerosis as a peculiar focal disease in the sense of localized osteodystrophia fibrosa which occurs in the labyrinthine capsule without regard to where the focus is or to whether or not it expresses itself clinically (by ankylosis of the stapes). Thus, in a series of 56 cases otosclerosis was discovered accidentally at autopsy at Lange's clinic in Leipzig. In none of these could the condition have been recognized clinically, either because the footplate of the stapes was not involved or because of otitis media. Absence of a family history of clinical otosclerosis, therefore, does not mean absence of histologic otosclerosis in the family. Weber states that clinical otosclerosis occurs in 31 per cent, or about one third, of the cases of histologic otosclerosis. The mendelian proportion of three sound to one diseased in recessively inherited characteristics is thus found, accounting for the assumption of some authors that otosclerosis is a recessive characteristic. Taking into account, however, that for every case of clinical otosclerosis in a family there are three cases of histologic otosclerosis, the theories of heredity based on family histories are seen to be false. While a satisfactory study of the inheritance of otosclerosis will not be made until all members of a family are examined histologically, Weber regards otosclerosis as a dominantly inherited condition.

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2. Weber, M.: Zur Frage des Erbganges der Otosklerose, Erbbl. f. d. Hals-, Nasen- u. Ohrenarzt, no. 1-3, November 1936, p. 3.

A genetic study of an otosclerotic family is reported by H. F. Schick and M. A. Goldstein,[3] in which the deafness was traced back for four generations. This study is of particular interest because otosclerosis appeared in every generation, and when defective hearing was present on both sides of the family not only was there an increase in the incidence but the deafness seemed to come at an earlier age. One child in the fourth generation was deaf at the age of 3 years, owing, the authors suggested, to otosclerosis, though she was too young for accurate tests of hearing. The assumption that she had otosclerosis, however, seems less probable than the assumption that she lost her hearing from some intercurrent infection or was congenitally deaf, since clinical otosclerosis is rare in childhood.

A study of heredity in 408 cases of otosclerosis from the Leningrad Institute for the Throat, Nose and Ear is reported by T. P. Schtschelkanowa.[4] He found a family history of deafness in only 38 per cent of the patients and concludes that otosclerosis is not hereditary. The low incidence of a family history among these patients compares to a family history in 72 per cent of the cases of otosclerosis from the Washington League for the Hard of Hearing studied by my associates and me in 1933. The difference is probably explained by the different social and economic status of the two groups, since poorer people, as a rule, know far less about their relatives than do the well-to-do. The conclusion that otosclerosis is not hereditary is contrary to the established facts and to the view now held almost universally that the single unquestioned etiologic factor in otosclerosis is heredity.

An interesting pathologic study of a case of otosclerosis is reported by A. A. Gray.[5] A physician with a deafened sister who could hear better in noisy than in quiet surroundings began to lose his hearing between 40 and 50 years of age. Except for some discharge from the left ear in childhood, there was no previous history of aural disease. At 53 he retired from practice because of deafness. At 56 he could carry on a conversation, but with some difficulty. At 66 he used a speaking tube, but in the following year, after a severe illness, he became completely deaf for speech. At 71 he could not hear the middle and lower tuning forks, but he could hear notes above 2,048 double vibrations produced with the Galton whistle. At 86 he died, having willed that his ears should be examined after death.

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3. Schick, H. F., and Goldstein, M. A.: Genetics in Otosclerosis, Laryngoscope **46**:731 (Oct.) 1936.

4. Schtschelkanowa, T. P.: Zur Frage über die Erblichkeit der Otosklerose, Arch. sovet. Otol., no. 1, 1936, p. 41.

5. Gray, A. A.: The Pathological Changes Found in a Case of Otosclerosis, Acta oto-laryng. **24**:96, 1936.

Examination showed a typical, sharply demarcated area of osteoporosis anterior to the left oval window, extending onto the footplate of the stapes and to the anterior border of the oval window, with ankylosis of the stapes. The cochlea, including the organ of Corti, the cochlear nerve and the spiral ganglion, showed no changes. Even more surprising in view of the profound bilateral deafness was the absence of an otosclerotic focus in the right ear or of any pathologic changes in the middle or inner ear which could account for the deafness. The author suggests that the unusual finding of otosclerosis on only one side (1 case was reported by Manasse) might have been due to the suppurative process in childhood in the affected ear, which disturbed the local vasomotor system in such a way as to bring about the osteoporotic change. The profound bilateral deafness could be explained only by some central lesion, but as yet the brain and the medulla of this patient had not been examined.

Further studies of the two generalized diseases of the bone which are frequently complicated by the clinical picture of otosclerosis—osteogenesis imperfecta (fragilitas ossium) and osteitis deformans (Paget's disease)—are reported in the literature for 1936.

G. Guillain and M. Aubry [6] studied the labyrinth in cases of Paget's disease macroscopically, histologically, roentgenologically and clinically. Macroscopically, a rotation of the axis of the labyrinth may occur, so that one can look into the internal auditory meatus from above. This is characteristic of Paget's disease according to O. Mayer. Microscopically, the labyrinthine capsule is destroyed and replaced by new bone with a varying degree of atrophy of the nerve elements in the labyrinth. The roentgenogram shows blurring of the contours of the petrous pyramid with hazy spots in the bones and in adjacent bones. Clinically, in the early stage of Paget's disease of the labyrinth, there is a mixed type of deafness with prolonged bone conduction and elevation of the lower tone limit and also a lowering of the upper tone limit. Later, there is increasing labyrinthine involvement, but the prolonged bone conduction persists. Characteristic of Paget's disease are the steady progress of the deafness and the marked labyrinthine deafness with prolonged bone conduction.

H. Brunner and E. Grabscheid [7] describe in detail the histologic observations in osteitis deformans (Paget) of the petrous bone. No substantial enlargement of the petrous bone could be demonstrated. The pneumatic cell partitions showed rebuilding processes, and the cells

6. Guillain, G., and Aubry, M.: Le labyrinthe dans la maladie de Paget, Presse méd. **44**:889 (June 3) 1936.

7. Brunner, H., and Grabscheid, E.: Zur Kenntnis der Osteitis deformans (Paget) der Schädelbasis: I. Das Schläfenbein, Virchows Arch. f. path. Anat. **298**:195, 1936.

contained cellular exudate, with inflammatory changes in the mucoperiosteum. The labyrinthine capsule was thickened, and the internal auditory meatus was narrowed. The membranous inner ear showed marked atrophy of the whole nerve ganglion apparatus, and in addition there was serous labyrinthitis. The authors believe that Paget's disease is a chronic inflammatory disease of the bone of unknown etiology. The serous labyrinthitis is part of this chronic inflammation and occurs when the rebuilding process reaches the endosteum of the labyrinth. The 68 year old patient whose temporal bone was studied complained shortly before death of attacks of Ménière's disease, doubtless due to the serous labyrinthitis.

J. R. Lindsay and H. B. Perlman [8] found involvement of the skull in 7 of 25 patients with Paget's disease, and 4 of these had impaired hearing. Two showed a negative Rinne and a prolonged Schwabach reaction without sufficient changes in the middle ear to account for this conduction deafness; so presumably the osseous changes of Paget's disease around the oval window had caused fixation of the stapes. In 1 patient, with a pink glow from the promontory and a negative Rinne reaction, the Rinne reaction became positive as the pink glow receded. All 4 patients showed considerable labyrinthine deafness, as evidenced by loss of perception for the upper tones and a lowering of the upper tone limit. The authors consider this marked labyrinthine involvement to be characteristic of Paget's disease.

V. Tanturi [9] presents a series of 7 patients with fragilitas ossium, blue scleras and deafness. While the brittleness of the bones is the most prominent symptom, the deafness may be present alone, the family history of frequent fractures showing that the condition belongs to this syndrome. The deafness is due to otosclerosis, as has been histologically determined in several cases reported in the literature. The etiology of fragilitas ossium is not clear. In addition to the brittle bones, blue scleras and deafness, brittle hair and nails, marked disease of the teeth and poorly developed intelligence are often associated with this condition.

T. Ritchie Rodger [10] states that the most common symptom of the syndrome is the blue scleras, 60 per cent of the patients with this condition having an associated liability to fracture and 60 per cent associated deafness. The condition is inherited as a dominant characteristic with no sex preference. Clinically and histologically the deafness is

8. Lindsay, J. R., and Perlman, H. B.: Paget's Disease and Deafness, Arch. Otolaryng. **23**:580 (May) 1936.

9. Tanturi, V.: Osteopatirosi, steri bleu e disturbi auricolai, Rassegna ital. d'ottal. **9**:98, 1935.

10. Rodger, T. Ritchie: Otosclerosis Associated with Blue Sclerotics and Fragilitas Ossium, Proc. Roy. Soc. Med. **29**:1107 (July) 1936.

indistinguishable from otosclerosis. The condition of the eyes and the bones is present from birth. The deafness, however, does not begin until the third decade. In discussing Rodger's paper, Nager brought out that the pathologic picture of the otosclerotic focus found in these cases is different from the generalized condition of the bones, which was a defective functioning of the osteoblasts. Thus, two different diseases of the bone are found associated in these patients.

Three cases of deafness following the prolonged administration of quinine, 2 of the patients showing a positive reaction to the tests for otosclerosis, are reported by L. F. Day.[11] Because he has noted in patients who are receiving large doses of quinine a reddish blush posterior to the umbo, which he ascribes to local hyperemia, the author believes that in his cases the otosclerosis was due to the quinine, which caused hyperemia leading to the absorption of circumscribed areas of bone in the labyrinthine capsule, with the replacement by spongy bone. This conclusion hardly seems justified on the evidence presented.

The literature for 1936 contains the usual number of reports of attempts to connect otosclerosis with metabolic, endocrine or vitamin disturbances.

From France come two reports purporting to show the connection of otosclerosis with the parathyroid glands. Because alterations in calcium metabolism in otosclerosis have been reported in the literature and because the parathyroid glands regulate the blood calcium level, J. M. Alonso and A. Chiarino [12] removed one parathyroid gland or tied the terminal branches of the inferior thyroid artery on one side in 7 patients with otosclerosis and obtained a slight improvement in hearing in 6 of the 7. Having found a relation between otosclerosis and tuberculosis, the same authors then used an antituberculosis vaccine in some cases of otosclerosis and obtained improvement in hearing. Finally, they tried calcium and irradiated ergosterol in treating otosclerosis, and again improvement in hearing resulted.

A. Malherbe,[13] assuming that otosclerosis is due to a lack of parathyroid secretion, obtained a striking improvement in hearing after the administration of parathyroid extract to patients with otosclerosis. This author, therefore, neatly contradicts Alonso and Chiarino, who improved the hearing by removing one parathyroid gland!

Equally unconvincing is a paper by E. Charschak,[14] who improved the hearing in several cases of otosclerosis, with disappearance of

11. Day, L. F.: Quinine and Otosclerosis, Malayan M. J. **11**:53 (March) 1936.

12. Alonso, J. M., and Chiarino, A.: Contribution à l'étude de la pathogénie et du traitement de l'otosclérose, Rev. de laryng. **57**:1106 (Dec.) 136.

13. Malherbe, A.: Oreille et parathyroïde, Presse méd. **44**:1484 (Sept. 23) 1936.

14. Charschak, E.: Ein Versuch, die Otosclerose mit bestrahlten Hefen zu behandeln, Acta oto-laryng. **23**:445, 1936.

tinnitus, by the oral administration of irradiated brewers' yeast, thus combining vitamin B with vitamin D.

From the Leningrad Institute for the Throat, Nose and Ear, S. M. Scheschko [15] reports a noticeable diminution of the blood chlorides in all of 78 cases of otosclerosis studied. The author sees in this lowered chloride content an indication of chemical indolence of the cells, pointing to a diminished immunobiologic state of the organism which may serve as an etiologic basis for the development of otosclerosis. N. P. Belkina,[16] also from Russia, studied the potassium and calcium content of the blood in otosclerosis and found in 75 patients an elevation of the value for blood calcium. This hypercalcemia ran parallel to the stage of the process and reached its maximum in the second stage, that is, in the period of resorption of bone tissue. The potassium-calcium ratio was 1:5 instead of 1:7 to 1:9, as in normal blood. S. G. Rabinowitsch,[17] likewise Russian, determined the total value for blood nitrogen in 80 patients with otosclerosis. In all stages of the disease there was an elevation in the total nitrogen content of the blood serum, the lower level in these patients being higher than the upper level in normal persons. The more pronounced the picture of otosclerosis, the higher the values for total nitrogen. The author interprets his findings as evidence of a generalized metabolic disturbance. These three reports from the Russian literature are similar in that the authors obtained positive results in finding an abnormal blood chemistry in otosclerosis, whereas similar studies previously carried out elsewhere have generally given entirely negative results. Did these authors have a more accurate method of measuring the blood chlorides, calcium, potassium and nitrogen than previous workers, or did their desire to obtain positive findings outbalance their desire to determine the actual facts?

K. A. Drennova,[18] of the Leningrad Institute for the Throat, Nose and Ear, in an attempt to find a biochemical alteration in otosclerosis which might throw some light on its etiology, has followed an old superstition concerning the poisonous character of the blood of menstruating women. Other workers had previously shown that the blood, perspiration and saliva during the menses are more toxic than at other times, apparently because of an increased choline content. Having studied the venous blood, blood serum and menstrual blood of 7 normal

15. Scheschko, S. M.: Die Charakteristik des Chloranions der Blutes bei Kranken mit Otosklerose, Arch. sovet. otol., no. 1, 1936, p. 316.

16. Belkina, N. P.: Blutelektrolyten (Ca und K) bei Otosklerose, Arch. sovet. otol., no. 1, 1936, p. 303.

17. Rabinowitsch, S. G.: Niveau des allgemeinen Stickstoffs der Blutserums bei Otosklerose, Arch. sovet. otol., no. 1, 1936, p. 309.

18. Drennova, K. A.: Wirkung des Blutes von Otosklerosekranken auf des Leben der Pflanzen und Tiere, Monatschr. f. Ohrenh. **70**:826 (July) 1936.

women, 24 otosclerotic women and 7 otosclerotic men, the author concludes that the blood of otosclerotic persons is less toxic than that of normal persons when tadpoles, chrysanthemums or tritones are placed in various dilutions of these fluids. However, when one analyzes his results carefully, one finds that they are far from uniform, so that in some cases the blood of otosclerotic persons was more toxic than that of normal persons. Moreover, the individual results varied so widely that the author hardly seems justified in his conclusion that the blood of otosclerotic persons is less toxic than that of normal persons and that this difference can be explained by dysfunction of the adrenal glands (which regulate the level of choline) and that therefore otosclerosis is dependent on a general endocrine or trophic disturbance.

Another metabolic study of otosclerosis is reported by G. Orso,[19] from Hungary, who made 45 basal metabolic tests in 30 otosclerotic patients. Only 4 had a normal metabolism, the rest showing an elevation of from 15.3 per cent to 57.6 per cent, although only 2 of these had other symptoms of exophthalmic goiter. The tinnitus had no relation to the height of the metabolism, but the administration of colloidal iodine was effective, not only in restoring the metabolism to normal (in all but 3 cases) but in improving the tinnitus. An improvement in hearing occurred in 3 cases. The author believes that the elevated metabolism is primary in otosclerosis and that by the early use of iodine the condition can be arrested or improved. This surprising report is as hard to believe as the reports from Russia.

An even more fantastic-sounding paper is by N. D. Marejew,[20] of Russia, who made solutions of the petrous pyramid, cochlea and acoustic nerve and injected these into persons with otosclerosis and primary nerve deafness. He reports that the pyramid lysate improved ringing and whistling, whereas lysate of the cochlea and the acoustic nerve not only helped the tinnitus but improved the hearing!

The only report in the literature for 1936 on Gray's thyroxin treatment of otosclerosis was by M. A. Goldstein,[21] who used the technic and dosage described by Gray on 4 patients with otosclerosis. Two showed subjective and objective improvement, and 2 were not improved. G. Chubb [22] calls attention to the fact that Gray used no controls and when one control was accidentally present in that the untreated ear

19. Orso, G.: Grusdumsatzuntersuchungen bei Otosklerose, Orvosképźes **26**: 112, 1936.

20. Marejew, N. D.: Die ersten Versuche zur Behandlung der Otosklerose mittels Lysaten, Zhur. ushn., nos. i gorl. bol. **13**:395, 1936.

21. Goldstein, M. A.: Thyroxine in Treatment of Otosclerosis, Laryngoscope **46**:112 (Feb.) 1936.

22. Chubb, G.: The Problem of Otosclerosis, Brit. M. J. **2**:1192 (Dec. 12) 1936.

showed greater improvement than the treated ear, Gray attributed this to a "crossed reflex!" Chubb states that subsequent investigation has entirely failed to justify Gray's expectations. He also calls attention to the ease with which otosclerotic persons can be convinced that they hear better, irrespective of the treatment, which explains the continued use of the Zünd-Burguet treatment in some quarters, although such men as Professor Nager and Fraser have never seen any results from this method.

Contrasting with these unconvincing reports of improved hearing in otosclerosis from various medical treatments are two interesting and important papers published in 1936 on the surgical treatment of otosclerosis.

G. Holmgren [23] calls attention to the fact that none of the medical treatments for otosclerosis have given significant results, if one may speaks of results at all. On the contrary, every operator who has worked on the problem of the surgical relief of otosclerosis has obtained an instantaneous apparent improvement in hearing as soon as a sound fistula into the labyrinth was established, irrespective of at what point. This first occurred when Kessel in 1876 extracted the ankylosed stapes and again in 1897 when Passow made a sound fistula into the promontory with a trephine. The improvement in hearing, however, was not great and lasted only a few days. General opposition arose against these operations as dangerous to the life of the patient as well as to the hearing and as without permanent results, and not until ten years later was the question revived by Bárány, who made a sound fistula into the posterior vertical canal, but again the improvement in hearing lasted only a few days. In 1913 Jenkins made a fistula into the horizontal canal, which he covered with a skin graft, obtaining immediate improvement in hearing in 2 cases of otosclerosis, but when 1 patient became completely deaf in a short time and the other soon lost the improvement in hearing, he abandoned the operation.

Assuming that a lasting improvement in hearing would result if one covered the sound fistula at operation with a yielding membrane, Holmgren in 1917 made a fistula on the upper curve of the anterior vertical canal, exposing this to the dura. The immediate result was good but of brief duration. Bárány then made a fistula into the horizontal canal, covered it with a pad of fat previously transplanted into the mastoid cavity and obtained an improvement in hearing lasting fourteen days.

In 1920 Holmgren, using magnifying optical instruments, made an opening into the promontory, which he covered with the mucoperiosteum

23. Holmgren, G.: Zur Chirurgie der Otosklerose, Acta oto-laryng. **24**:271, 1936.

which he had previously elevated; but again the result was only temporary. Two years later he used the same technic on the horizontal semicircular canal, with permanent improvement in hearing in some cases, though the improvement was never great. Selecting patients who had otosclerosis with prolonged bone conduction, he operated on a total of 35 over fifteen years with good, in fact often very good, hearing immediately, but then a loss, so that the final result was never a pronounced improvement. However, in many of these cases the operation seems to have arrested the progress of the disease, since the poorer hearing ear was always selected for surgical treatment and now is, in many cases, the better ear.

In 1924 Sourdille began working on this question with enthusiasm, with results and with a technical skill which Holmgren describes as brilliant. Encouraged by this, Holmgren further refined his technic. Using local anesthesia, he makes a wide antrotomy and every vestige of bleeding from the bone is stopped by bone wax. The saccus endolymphaticus is then exposed, and a fistula is made into the horizontal and vertical semicircular canals with a chisel. As soon as a fistula is established, the hearing improves considerably—conversation can be heard at about 10 meters. The fistulas are made from 4 to 5 mm. long, and the endosteum is removed without injury to the membranous labyrinth. A prosthesis of fat is then placed over the fistula, the inner wall of the antrum being first covered with gold leaf to prevent adhesion to the membranous labyrinth. The incision is closed with Michel clips.

Since February 1935, 34 patients have been operated on with different modifications of this technic, with no serious complications and no deaths. At first the patients suffered from vertigo, which disappeared after a month or less. The patients who before operation suffered from tinnitus reported that this disappeared entirely or almost entirely with the establishment of the sound fistula. When the improvement in hearing disappears, the tinnitus returns.

The improvement in hearing is constant and surprising at the first escape of labyrinthine fluid, as observed through the microscope. A few hours after operation, conversation is heard at 20, 30 or even 40 meters. In the healing period the hearing again becomes poorer; in some cases it goes back to its previous level, and in 2 cases it even sank lower. In the majority of cases a more or less permanent improvement in hearing is attained, which in certain cases even gradually increases. Of the last 13 persons operated on, 11 show a clear improvement; 8 of them show considerable improvement.

The author emphasizes the need of several years' observation before one is entitled to speak of a final improvement in hearing. He also stresses the well known variations in hearing in otosclerosis with

changes in weather, nervousness, etc. All his patients are examined by two persons, with hearing tests in a sound-proof and in a normally noisy room and with audiometric studies. All in all, one is favorably impressed by the author's conservatism in evaluating his results.

A second interesting paper on the surgical treatment of otosclerosis is by Sourdille.[24] Stimulated by a trip to Stockholm and Upsala in in 1924, where he observed Holmgren's and Bárány's technic of microscopic surgical treatment of the ear, he undertook to transform the conducting mechanism so that the vibrations of the drum membrane would be transferred to the fistula in the horizontal canal. The technic which he has finally evolved in the course of 323 operations on 109 ears requires three operative interventions separated from one another by from three to four months. First, a modified radical mastoidectomy is performed, with removal of the bridge but careful preservation of the drum membrane and the ossicles. At the second operation, a plastic operation on the pars flaccida and the head of the malleus allows the sound vibrations to be carried to the region of the horizontal semicircular canal. At the third operation the fistula into the horizontal canal is made and the pars flaccida previously operated on is placed over this. The same improvement in hearing observed by Holmgren as soon as the perilabyrinthine space is exposed to the free air is reported by this author. As soon as a cicatricial membrane covers this fistula, the hearing diminishes. By altering the tympanic conducting apparatus and covering the fistula by a thin membrane, the author largely avoids this secondary loss of hearing. The operative results have been 74 successes from 100 operations, but with improving technic the results are better.

In reading this paper one is impressed by the technical difficulties of the series of operations and by the immense patience required not only by the surgeon but by the patient, who must spend the better part of a year before the result is obtained. This paper is, perhaps, less convincing than Holmgren's, since there is no mention of complications or mortality and no report of cases. The end-results of this prolonged technic, of three successive operations, are apparently better than those after Holmgren's single operation.

An interesting paper by E. P. Fowler [25] suggests a new method for the early detection of otosclerosis. Certain deafened persons appear to hear loud sounds better than their threshold audition would imply. The author suggests testing the acuity of hearing for loud sounds as

24. Sourdille, M. M.: Traitement chirurgical de l'otospongiose, Oto-rhino-laryng. internat. **19**:798, 1935.

25. Fowler, E. P.: Method for Early Detection of Otosclerosis, Arch. Otolaryng. **24**:731 (Dec.) 1936.

well as for minimum audibility at threshold, as is usually done. By using a tone considerably above threshold and throwing it alternately into the two ears, varying the intensity until it sounds equally loud in the two ears, the acuity of hearing for loud sounds can be accurately compared. The author calls this the alternate binaural loudness balance. This entirely new method of testing hearing acuity yielded interesting results. In cases of conduction deafness it was found that if there was a difference in the threshold between the two ears of 20 db. for a given tone, at 30 db. above threshold there was still a difference of 20 db., as determined by the alternate binaural loudness balance. In nerve deafness, on the other hand, the higher the sound was above threshold, the less the difference between the two ears, until at 60 db. above threshold a given tone may sound equally loud in the two ears, whereas at threshold there was a difference of 20 db. In a few persons with normal or nearly normal hearing but with a family history of otosclerosis, the author found little or no difference between the two ears at threshold but a distinct difference at higher intensities. He suggests that this is due to the "hobble" effect of early changes in the annular ligament of one ear, too slight to interfere with vibrations of low intensity but enough to cut down the large excursions produced by loud sounds.

LABYRINTHINE DEAFNESS

Combining their pathologic material, O. Mayer and J. S. Fraser [26] report the histologic changes in the ear in late congenital syphilis. The authors point out that congenital syphilis of the ear, besides being associated with interstitial keratitis and dental deformities, usually appears between the tenth and the twentieth year, generally in females, and once started the deafness progresses rapidly, becoming pronounced overnight in some cases. The Wassermann reaction is usually negative.

The first patient, a man of 20, had had normal hearing until the age of 4 years, when a discharge from the ears began after an attack of measles, with slight and gradually increasing deafness. At 10 years interstitial keratitis and ulceration of the pharynx occurred, and there ensued a rapid loss of hearing, reaching almost complete deafness within a year. Tinnitus and giddiness were present. The Wassermann reaction was negative. Histologic examination showed a gumma of the posterior meatal wall on each side, swollen mucosa in the middle ears, osteomyelitis of the malleus and incus, extensive chronic gummatous osteomyelitis of the labyrinthine capsule, with giant cells and necrosed particles of bone, extreme degeneration of Corti's organ and gummas in the spiral ganglion.

The second patient, a woman of 35, had been in good health until she was 15, when progressive disease of the joints developed, which finally made her bedridden. Complete deafness developed at the onset of the trouble with the joints, and difficulty with the vision developed ten years later. Histologic study showed bony

26. Mayer, O., and Fraser, J. S.: Pathological Changes in Ear in Late Congenital Syphilis, J. Laryng. & Otol. **51**:683 (Nov.) 1936.

ankylosis of the malleus to the incus. The cochlea showed serous labyrinthitis, with coagulation of the endolymph and perilymph and slight formation of new connective tissue in the perilymphatic spaces. The organ of Corti, the stria vascularis and the spiral ganglion were atrophic. The semicircular canals showed extensive gummatous periostitis.

The third patient, a man of 35, suddenly became deaf at the age of 10 years and manifested a rolling gait. Examination at that time showed no hearing and no response to rotation. Histologically, the middle ears showed only slight changes. The labyrinthine capsule was largely replaced by lamellar bone, the organ of Corti was highly atrophied and the semicircular canals exhibited profuse subperiosteal infiltration characteristic of syphilis.

The fourth patient, a woman of 75, could not hear the spoken voice but could hear the low-pitched forks and whistling well. Histologically, the middle ears were normal; the cochlea showed small miliary gummas in the endosteum of the spiral ligament and the scala tympani, but the organ of Corti was in good condition. The labyrinthine capsule was changed to lamellar bone in places, with the subperiosteal chronic periostitis of the semicircular canals characteristic of syphilis.

The fifth patient, a woman of 56, had become deaf at 39 and blind at 46, and osteomalacia kept her in bed from the age of 50. Histologically, the labyrinthine capsule showed granulation tissue at the border of the periosteal capsule, which had eaten into the bone and invaded the labyrinth. Erosion everywhere was replaced by formation of new bone or by a loose spongy mass of granulation tissue. There was a serous labyrinthitis, with atrophy of Corti's organ.

In a study of the ears of 207 syphilitic persons, S. Maruyama and T. Tarasi [27] found that involvement of the inner ear in the early stage of syphilis is accompanied by a positive serologic reaction but in latent syphilis there are more cases in which there is a negative serologic reaction. In the early stage of syphilis there is no relation between positive findings in the spinal fluid and involvement of the inner ear, whereas in the late stages positive findings in the spinal fluid are frequently associated with changes in the inner ear. Syphilitic deafness occurs earlier than syphilitic aortitis. In most cases of syphilitic deafness the earlier treatment was defective or absent; between the untreated and the incompletely treated patients there was little difference in the incidence of deafness. Adequate treatment soon after the infection can protect the inner ear from syphilitic disease.

The German literature for 1936 is replete with articles on the diagnosis of hereditary deafness, the result, of course, of the new law providing for the sterilization of the hereditarily deaf.

Langenbeck [28] brings forth more evidence in favor of his law of symmetry in hereditary deafness. He found that four fifths of 140 persons with hereditary deafness showed a picture of symmetrical hearing. On the basis of this, the author believes that when a sporadic

27. Maruyama, S., and Tarasi, T.: Ueber die Functionsprüfrung des Gehörorgan bei Syphilitikem, Hihuto-Hitunyo **4**:29, 1936.

28. Langenbeck, B.: Symmetrische Hörreste und Erblichkeit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:486, 1936.

case of deaf-mutism occurs in a family otherwise free from deafness and symmetrical remnants of hearing are found, one is justified in assuming that the deafness is hereditary.

E. Wirth [29] confirms Langenbeck's law of symmetry on the basis of a study of 100 deaf-mutes with normal drum membranes. Of these, 76 in all probability inherited the condition and 24 probably or certainly acquired it. Of the former, 39 had no family history of deafness and 37 had a family history of deafness. Since 4 of the 37 had asymmetrical remnants of hearing, the finding of asymmetry in doubtful cases is not valid evidence against heredity. Of the patients who had certainly acquired deafness (12 cases) half had remnants of hearing, and these all showed different hearing curves for the two ears. Thus, asymmetry in general speaks for acquired deafness. Of the 39 patients who probably had inherited deafness but who did not have a family history of deafness, there were 12 with symmetrical remnants of hearing and 18 with asymmetrical remnants. Of the 37 patients with inherited deafness who had a family history of deafness, there were 17 with symmetrical remnants and only 4 with asymmetrical remnants. The author concludes that symmetrical remnants are more often present in inherited deafness with a history of deafness in the family. (Why did he not conclude that in many cases in which there was no family history the deafness was not inherited?)

The result of the vestibular tests showed that disturbed responses (usually with complete loss) are frequent in hereditary deafness and acquired deafness and normal responses also occur in both types.

Absolute proof of hereditary deafness depends on the finding of bilaterally symmetrical hearing curves and deaf-mute siblings.

M. Schwartz [30] differentiates between dominant congenital hereditary degenerative inner ear deafness (easily diagnosed from the family history) and sporadic or recessive deaf-mutism. In the latter the family history must be searched diligently, and if a single other case is found the diagnosis of hereditary deafness is justified and the rejection of sterilization can occur only when an acquired cause for the deafness is demonstrable.

W. Lange [31] examined histologically the ears of 14 deaf-mutes and found in 7 incontestable residues of labyrinthine inflammation. One patient showed a malformation of the membranous labyrinth. One

29. Wirth, E.: Erbliche Belastung, Seitenverteilung der Hörreste und Vestibulariserregbarkeit bei Taubstummen und ihre Bedeutung für Erbbegutachtung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:212, 1936.

30. Schwartz, M.: Die Erkennung der erblichen Taubheit, Öff. Gesdh. dienst **2**:44, 1936.

31. Lange, W.: Die histologische Feststellung von erblichen Taubheit und Schwerhörigkeit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:1, 1936.

showed normal labyrinths. The remaining 5 showed changes in the endolymphatic spaces, in the spiral ganglion and in the nerves, resembling closely the posthydropic neuro-epithelial degeneration described by Wittmaack. While this degeneration may be the result of some outside influence (4 of the 5 patients had had a previous suppurative otitis media), the author believes that weakness of these structures, making them susceptible to injury is inherited and that, therefore, heredity cannot be rejected.

W. Uffenorde [32] opposes the assumption of Lange that inherited sensitivity or weakness of the inner ear is to be blamed for the deafness, because it is more probable that a previous inflammation of the middle ear is the cause. Serofibrinous or purulent labyrinthitis occurs much more frequently in children in association with otitis media than in adults, and this induced labyrinthine inflammation in small children is important as a cause of deaf-mutism. The author also states that symmetrical remnants of hearing occur in acquired deafness, so that Langenbeck's law is not sufficient evidence. The proof of heredity must lie in the family history alone. The author concludes: "We have every ground to exercise the greatest care and caution, and with all readiness to support the law for eliminating hereditary disease we must keep our professional obligation to the patient as well as to our people."

During 1936 there appeared many reports of toxic inner ear deafness from drugs or bacteria.

Having previously shown that procaine hydrochloride, quinine, alcohol and hydrochloric acid are capable of passing from the normal middle ear to the endings of the vestibular nerve and there producing functional changes, E. L. Ross and R. W. Rawson [33] injected bacterial toxins into the middle ears of dogs. The function of the labyrinth was tested by the rotation test and the caloric test and the hearing by the conditioned reflex response. The toxins of staphylococci, pneumococci, hemolytic streptococci and tetanus bacilli produced no change when left in the normal middle ear for half an hour. The toxin of scarlet fever reduced the nystagmus response slightly in 3 of 4 dogs. The toxin of diphtheria, on the other hand resulted in a marked reduction of nystagmus, with failure of every animal to respond to loud sounds.

A histologic study was made by W. P. Covell [34] of the changes in the cells of the organ of Corti and the spiral ganglion after the administration of quinine and salicylates to guinea-pigs. The mitochondria

32. Uffenorde, W.: Zur Beurteilung der erblichen Taubheit, Erbbl. f. d. Hals-, Nasen- u. Ohrenarzt, no. 4, December 1936, p. 43.

33. Ross, E. L., and Rawson, R. W.: Effects of Bacterial Toxins in the Middle Ear, Arch. Otolaryng. **24**:51 (July) 1936.

34. Covell, W. P.: A Cytologic Study of the Effects of Drugs on the Cochlea. Arch. Otolaryng. **23**:632 (June) 1936.

of the hair cells, the spiral ganglion cells and the cells of the stria vascularis were found to show distinct changes when compared with those of the normal controls. These changes are most marked in the basal coil of the cochlea but may involve the apical coils when the drug has been administered over a longer period. The two drugs produced slightly different effects, quinine causing more pronounced changes in the cells of the organ of Corti while salicylates produced a greater degree of change in the cells of the spiral ganglion. Of especial interest in view of Taylor's theory that quinine is a frequent cause for congenital deafness was the finding that the cochleas of the fetus showed more marked changes than did those of the mother when these drugs were given to pregnant guinea-pigs.

K. Bernfeld [35] reports a case of deafness from chenopodium poisoning. A woman of 50 accidentally took 5 Gm. (5 times the normal dose) of oil of chenopodium. Within ten minutes she felt ill and vomited; this was followed by convulsions and then by coma. Twenty minutes later she awakened with tinnitus and vertigo and could hear with neither ear. Eight days later, when examined, she could hear the voice at the ear. Treatment consisting of injections of pilocarpine, electrical treatments and sweating were followed by an improvement in hearing after a few days. Sixteen days after the onset of the trouble the voice was heard at a distance of 70 cm. from each ear.

An extraordinary case of labyrinthine deafness due to a tapeworm is reported by E. di Lauro.[36] Severe inner ear deafness was associated with tinnitus and vertigo. Treatments were fruitless. One day the head of the tapeworm was passed, with instantaneous cessation of all the aural symptoms!

E. Ruttin [37] found a trace of arsenic in the paint on the walls of the home of a 30 year old patient with inner ear deafness on the left side with vertigo, and because no other cause for the deafness could be found he attributed it to arsenic poisoning.

M. Oyamada [38] injected the toxin of Bacillus pyocyaneus into the middle ears of guinea-pigs and obtained serous labyrinthitis, with marked degeneration of the nerve apparatus of the cochlea and the spiral ganglion. Injection of the toxin subcutaneously resulted in

35. Bernfeld, K.: Zur Symptomatologie der Neuropathia acustica toxica nach Chenopodiumölvergiftung, Monatschr. f. Ohrenh. **70**:353 (March) 1936.

36. di Lauro, E.: Turbe labirintiche da "Taenia solium," Valsalva **12**:437 (Sept.) 1936.

37. Ruttin, E.: Ueber Schädigung des Gehörorgans durch arsenhaltigen Maueranstrich, Acta oto-laryng. **23**:414, 1936.

38. Oyamada, M.: Experimentelle Untersuchung über den Einfluss des Bacillus pyocyaneus und dessen Toxin auf das Gehörorgan, Fukuoka acta med. (abstr. sect.) **29**:97 (Aug.) 1936.

similar marked degeneration of the nerve apparatus in the cochlea. The changes in the vestibule and the semicircular canals were slight. The author concludes that the toxin of B. pyocyaneus has an especial affinity for the nerve elements of the cochlea.

In a discussion of toxic neuritis of the acoustic nerve, Z. Szolnoky [39] states that it is the cause of inner ear deafness in a high percentage of cases, the result either of exogenous toxins (alcohol, nicotine, quinine, etc.) or of endogenous toxins from bacterial infection, gout or diabetes. The infections which can produce bacterial toxins injurious to the nerve of hearing are measles, scarlet fever, tuberculosis, influenza, mumps, herpes zoster oticus, acute and chronic suppuration of the bone and syphilis. In the therapy of toxic nerve deafness, removal of the etiologic factor is of first importance; then detoxication of the organism.

Of 74 diabetic persons, 43 were found by G. Jannulis and G. Dalijannis [40] to show acoustic phenomena. Of these, 14 had other conditions which could have caused the disturbance of hearing, leaving 29 whose defective hearing could be ascribed to the diabetes. All showed the reactions of nerve deafness, with a loss predominantly for high tones. In some cases the deafness appeared suddenly and was permanent; in these it was ascribed to central vascular changes. In others the deafness came on slowly and was usually reversible; in these it was believed to be due to toxic injury of the labyrinth. The changes in a few cases of gout were similar to those in diabetes.

Adolf Rainer [41] reports the case of a man of 26 who for fifteen years had had recurring multiple foci of osteomyelitis and who then acquired tinnitus and vertigo, with rapidly increasing nerve deafness leading in a little more than a year to complete deafness and absence of vestibular response. The author has found only 9 similar cases in the literature. He attributes the deafness to acoustic neuritis from the bacterial toxins associated with the osteomyelitis.

Several articles on the hearing in pregnancy appeared last year. L. Piccone [42] tested the hearing of 53 women at the end of pregnancy and some of them also at the onset and in the fourth month. In 30 per cent there was a diminution in hearing, in most cases due to disturbances of sound conduction (with elevation of the lower tone limit, an inconclusive to negative Rinne reaction and a prolonged Schwabach reaction,

39. Szolnoky, Z.: Neuritis nervi acustici durch Vergiftung, Orvosképzés **26**: 164, 1936.

40. Jannulis, G., and Dalijannis, G.: Diabetes und Gicht als Ursache von Cochlearis-schädigung, Monatschr. f. Ohrenh. **70**:1504 (Dec.) 1936.

41. Rainer, Adolf: Taubheit bei Osteomyelitis, Ztschr. f. Laryng., Rhin., Otol. **26**:344, 1935.

42. Piccone, L.: Contributo alle ricerche sulla funzione auditiva in gravidanza, Riv. d'ostet. e ginec. prat. **18**:389, 1936.

at times improved by catheterization) while 9 per cent of the patients showed a lesion of the inner ear. There were no cases of otosclerosis. The disturbances of hearing disappeared a few days after the birth, especially if the mother did not nurse the child. The author attributed these disturbances of hearing to local changes in circulation, pharyngotubal alterations, delayed assimilation and toxic manifestations of pregnancy due to renal dysfunction.

V. Sangiovanni [43] studied the ears of 100 pregnant women, 50 of whom showed albuminuria and elevation of blood pressure. He observed loss at the lower or upper tone limit and slight changes in the drum membranes, all of which returned to normal after the patients' delivery. These changes were no more marked in the patients with albuminuria and elevated blood pressure.

A. Rdzyminski [44] studied the hearing and vestibular reactions of 100 women during the last two months of pregnancy, one week after delivery and finally from two to three months after they stopped nursing. During pregnancy as well as during confinement, the author noted no disturbance of hearing. The vestibular apparatus, however, showed an increased irritability during pregnancy. The results of this study are directly at variance with those of Piccone and Sangiovanni, and although it is difficult to evaluate one study against another, this last investigation seems to have been carried out more carefully (tests were made on all cases at the three different periods) than the two preceding.

Occupational, or noise, deafness was studied by H. Minkim [45] in 65 workers in a noisy factory and in 40 other workers as a control. The changes of hearing in noise deafness and in old age are similar clinically and antamicopathologically, according to this author, except that in noise deafness there is an equal loss for c-4 and c-5, while in senile deafness the hearing for c-5 is affected much more than that for c-4. The Schwabach reaction is shortened in noise deafness, while the vestibular apparatus is little affected. The author's observation coincides with my own experience that noise deafness causes a dip in the audiogram in the region of c-4 and c-5, with better hearing above this region as well as for lower tones, whereas senile deafness is a progressive loss of the highest tones, gradually moving down the tone scale.

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43. Sangiovanni, V.: Sulla funzionalità uditiva in gravidanza, Arch. ital. di otol. **48**:217 (April) 1936.

44. Rdzyminski, A.: Ueber das Verhalten des Gehörs und des Gleichgewichtsorgans während der Schwangerschaft und der Entbindung, Polski przegl. otol. **12**:321, 1936.

45. Minkim, H.: Zur Frage der differentiellen Diagnostik zwischen den Veränderungen des Gehörs die durch Lärm und durch andere Ursachen bedingt sind, Zhur. ushn., nos. i gorl. bol. **13**:208, 1936.

For the prevention of occupational deafness, R. Perivitzschky and Lanez [46] studied the loudest frequencies that occur in a boiler works and found that these lie between 1,200 and 2,000 vibrations per second. A device was made to shut out these vibrations, consisting of two highly polished thin plates of metal (to provide a maximum of reflection of sound waves) separated for a space of from 1 to 2 mm. by a layer of rubber around the edges, with a layer of rubber on the surface which lies against the head to insure an air-tight fit. The device is comfortable. It reduces the distance at which the conversational voice can be heard to from 1 to 1.5 meters, and a whisper cannot be heard. It has been patented.

I [47] described a group of cases of Ménière's symptom complex in which the tests of hearing showed a perception type of deafness but with a greater loss of hearing for low tones than for the high tones and with distinct diplacusis, the tones being heard at a definitely higher pitch in the affected ear. This nerve deafness for low tones with diplacusis is explained as probably due to an inflammatory lesion in the cochlea, with weighing down of the vibrating membranes by edema or an adherent exudate, thus altering the vibratory response of this membrane. In all 3 patients a focus for infection was found, in 2 chronic tonsillitis and in the third chronic cystitis. In each patient the hearing returned to normal after removal of the focus, the diplacusis disappeared and there were no further attacks of vertigo.

The question of central deafness due to lesions in the brain has been, for the most part, an unexplored and unknown field. D. van Caneghem [48] reports a case of deafness of the left ear and spontaneous nystagmus associated with a choking of the left optic disk, facial paralysis on the left side and a positive Romberg reaction, all due to a tumor in the region of Rathke's pouch. In the course of several months the symptoms improved and the hearing for high tones returned sooner than that for low tones. The author attributes the improvement to a reduction of edema in the region of the lateral portion of the floor of the fourth ventricle.

N. Page [49] tested the hearing of 52 patients with tumor of the brain. Twenty growths were supratentorial, and 32, subtentorial. Of the latter, 18 were acoustic neuromas, and 3, tumors of the cerebellopontile angle.

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46. Perivitzschky, R., and Lanez: Gehörschutz für Arbeiten in Lärmbetrieben, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:178, 1936.

47. Shambaugh, G. E., Jr., in Medical Papers Dedicated to Henry A. Christian, Baltimore, Waverly Press, Inc., 1936, pp. 894-902.

48. van Caneghem, D.: Guérison d'un cas de surdité centrale complète. Contribution a l'étude de la physiologie pathologique des centres bulbo-protubérantiels du nerf cochléaire, Ann. d'oto-laryng., December 1936, p. 1221.

49. Page, N.: The Hearing in Patients with Intracranial Tumors: Report of Fifty-Two Cases, Bull. Neurol. Inst. New York **5**:287, 1936.

A distinct loss of hearing was found only with the acoustic neuromas and the tumors of the cerebellopontile angle.

A somewhat similar study was made by W. J. McNally and his associates.[50] In 6 cases of cerebellar tumor in which the lesion was confined to the cerebellum, there was little change in hearing. In 9 cases of cerebral tumor there was no deafness attributable to the lesion in the brain. In 4 cases of acoustic neuroma there was almost complete deafness on the involved side.

A most important contribution to the question of central deafness comes from H. Brunner,[51] who has made clinical and postmortem observations over fifteen years. He points out that the term "central deafness" is found frequently in neurologic and otologic literature, but the term is used loosely, the authors failing to make clear how central deafness is diagnosed and peripheral deafness excluded. H. Brunner undertakes to define sharply central deafness. He first considers the effects of increased intracranial pressure on the ear, and one must differentiate between general increased pressure and localized pressure.

Since one of the most prominent symptoms of general increased intracranial pressure is choking of the disk, the question at once arises: Is there an aural condition analogous to choking of the disk? To this one can answer definitely that changes in the inner ear analogous to choking of the disk have not been demonstrated.

The situation in regard to localized pressure in the brain, as in tumor of the posterior fossa, is different. A whole group of characteristic aural changes occur owing to (1) stasis hydrops of the inner ear or (2) central brain stem deafness. Stasis hydrops of the inner ear occurs as follows: A tumor of the posterior fossa, especially of the cerebellopontile angle can, when it reaches a certain size, compress the two principal veins which carry the blood from the inner ear into the superior petrosal sinus and the internal jugular vein. As a result, stasis occurs in the region of the inner ear, and transudate accumulates in the spaces of the inner ear. This transudate, when it has been present only a short time, can appear and disappear again without symptoms, but it can lead to a depression of the hearing. This finding occurs with cerebellar and occasionally with acoustic tumor.

Either through spontaneous lowering of pressure in the posterior fossa or by operative decompression, this transudate may disappear without leaving any injury to the inner ear. In these cases the depression of hearing will improve, and therefore one finds in cases of cerebellar and acoustic tumor variations in hearing during the clinical course,

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50. McNally, W. J.; Erickson, T.; Scott-Moncrieff, R., and Reeves, D. L.: Brain Tumors and Hearing, Ann. Otol., Rhin. & Laryng. **45**:797 (Sept.) 1936.

51. Brunner, H.: Pathologie und Klinik der Hörstörungen bei Hirntumoren, Wien. klin. Wchnschr. **49**:1037 (Aug. 21) 1936.

such as those which occur in multiple sclerosis and in certain types of syphilis of the inner ear.

This spontaneous disappearance of exudate from the inner ear does not always occur. The exudate can gradually become transformed into loose connective tissue and finally even into bone; one such case was observed years ago by Alexander. Of course, in these cases the depressed function of the inner ear cannot improve. Thus, stasis hydrops of the inner ear may occur without symptoms, with a temporary or with a permanent depression of hearing.

The tumors of the brain which frequently cause deafness are (1) tumor of the cerebellopontile angle, (2) tumor of the midbrain and (3) cerebellar tumor. Tumor of the cerebellopontile angle most often involves the acoustic nerve. The statement found in many textbooks that the cochlear symptoms are the first to appear does not hold, nor is complete deafness always found on the diseased side. The reactions to tests of hearing are variable and depend on the site of origin of the tumor, whether within the internal auditory meatus, where it springs as a rule from the vestibular nerve (lateral acoustic tumors), within the cisterna pontis lateralis (medial acoustic tumors) or, rarely, within the cochlea (multiple acoustic tumors).

An acoustic tumor may give no objective aural symptoms. This applies especially to medial acoustic tumor, but also to the lateral tumor when it is small and fails to fill the internal auditory meatus. However, when a lateral tumor lies under the slightest pressure in the internal meatus, it is likely to impair the function of the inner ear, soon causing complete or almost complete loss of nerve and ganglion cells in the inner ear, so that the impossiblity of any hearing can be confirmed microscopically. Clinically, associated with a lateral acoustic tumor there is a progressive loss of hearing of insidious onset, and when examined the patient is completely deaf or shows only a slight remnant of hearing on the diseased side. This deafness differs from real peripheral deafness in that the subjective tinnitus is usually not so prominent as that associated with otosclerosis or arteriosclerosis of the labyrinth, and the loss of function of the labyrinth, as shown by the caloric reaction, progresses parallel with the deafness. These two points are important in differentiating deafness associated with acoustic tumor from real peripheral deafness.

Occasionally, a unilateral acoustic tumor leads to bilateral deafness, but on the healthy side it almost never leads to complete deafness and only rarely is it combined with loss of labyrinthine function, differing in this respect from a bilateral acoustic tumor.

The hearing of a person with medial acoustic tumor is more complicated. The nerve ganglion apparatus in the inner ear is preserved to a considerable extent, even in the presence of a large tumor, as con-

trasted with the condition associated with a lateral acoustic tumor, already discussed. On the other hand, one frequently finds well developed stasis hydrops. Accordingly, tinnitus (due to atrophy of the nerves and ganglions in the inner ear) occurs late in these cases, often much later than labyrinthine vertigo. The hearing may also be considerable, even in the presence of a large tumor, and with this the caloric reaction is usually present. It is in association with deafness due to stasis hydrops that variations in the intensity of the deafness occur, with rarely an improvement in hearing after operation.

Deafness due to tumor of the midbrain is a slightly explored field. The secondary paths of the auditory nerves lie here. Siebenmann in 1896 first pointed out that disease in this region may lead to deafness. In his case there was a large gliosarcoma of the pineal gland, which had infiltrated the midbrain. Microscopically, the primary cochlear nuclei and the inner and middle ears were normal. The deafness was ascribed by Siebenmann to interruption of the secondary auditory pathways in the midbrain. He named this midbrain deafness.

Since this classic study, knowledge of midbrain deafness has not progressed. In three of the author's patients with less extensive tumor he found changes in the inner ear such as occur in the presence of acoustic tumor, which showed that the deafness was not purely from the midbrain.

Clinically, midbrain deafness can be unilateral or bilateral; if unilateral, it is due to interruption of the contralateral auditory tracts. As a prodromal symptom of this deafness, Siebenmann observed a marked shortening of bone conduction. The deafness occurs late, much later than that due to acoustic tumor. Being a late symptom, it is of unfavorable prognostic significance. The loss of hearing, once begun, shows a rapid, almost apoplectiform progression, so that only a trace of hearing can be found at examination. Of special help in differentiating it from acoustic tumor is the fact that midbrain deafness is accompanied, as a rule, by overactivity of the labyrinth.

Deafness associated with cerebellar tumor is rare; it is present in only about 16 per cent of the cases. This deafness can occur pathologically in three ways: 1. It may occur with tumor combined with meningeal disease in which the meningeal disease extends along the internal auditory meatus or perhaps along the cochlear aqueduct into the internal ear, leading to disease of the inner ear and deafness, like that associated with metastatic tumor or tuberculosis. The deafness is usually bilateral and involves the labyrinth as well as the cochlea. 2. Stasis hydrops of the inner ear may occur, with a fairly acute onset of deafness, without prodromal tinnitus, which may reach a considerable degree rapidly. It is characteristic, as already mentioned, that this type of deafness shows spontaneous variations and may improve after operation.

3. Deafness may be produced by invasion of the brain stem, leading to loss of labyrinthine function preceding loss of cochlear function, this loss being bilateral. Brain stem deafness thus differs from that arising from an acoustic tumor in being preceded by a loss of labyrinthine function and by bilateral deafness. Brain stem deafness differs from midbrain deafness in the loss of labyrinthine activity and in the presence of simultaneous pontile and medullary symptoms. It is similar to midbrain deafness in its late appearance and relatively rapid progression.

	Time of Appearance	Aural Symptoms	Course	Labyrinth
Lateral acoustic tumor	Early symptoms	Same side as tumor	Gradually progressive	Inactive
Medial acoustic tumor	Late symptoms	Same side as tumor	Gradually progressive	Decreased activity or inactive
Midbrain deafness.....	Late symptoms	Bilateral or on normal side	Rapidly progressive	Overactive
Brain stem deafness....	Late symptoms	Bilateral	Rapidly progressive	Inactive
Stasis hydrops.....	Late symptoms	Same side as tumor	Variations in intensity	Normal or decreased activity

Deafness arising from tumor in the temporal lobe has not been established in any case, and this is surprising, since the hearing center is located in this area.

A table differentiating the various types of central deafness has been drawn up by Brunner, though he takes pains to point out that while these broad generalizations usually hold true there are, of course, exceptions.

Abstracts from Current Literature

Ear

VARIATIONS OF THE CRANIAL VENOUS SINUSES IN THE REGION OF THE TORCULAR HEROPHILI. BARNES WOODHALL, Arch. Surg. **33**:297 (Aug.) 1936.

The clinical course, studies on the dynamics of the spinal fluid and the pathologic changes are recorded in a case in which a completely positive but false unilateral Queckenstedt reaction was exhibited. This type of irregularity in the Queckenstedt reaction, when applied in the diagnosis of thrombosis of the lateral sinus, may be due either to external pressure on the wall of the sinus or to a major abnormality of the sinuses, as illustrated in this case. Such an anatomic abnormality has been reported in 4 per cent of cranial sinus patterns. Two other types of irregularity in the Queckenstedt reaction, the unilateral false negative and the bilateral false positive reaction, are discussed. They may have a common origin in channels of collateral circulation, either preexistent in the second type or brought into play in the presence of thrombosis in the first.

GRANT, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

NEURALGIAS AND EAR SYMPTOMS, ASSOCIATED WITH DISTURBED FUNCTION OF THE TEMPOROMANDIBULAR JOINT. JAMES B. COSTEN, J. A. M. A. **107**:252 (July 25) 1936.

Costen, in a review of a large group of cases in which headache and aural symptoms were shown to be partially or altogether due to disturbed function of the mandibular joint, shows this to be a common factor. The pain and involvement of the ear are similar in every respect to those in some commonly known disorders of the eye, sinus and ear. Eighty-five patients fell into the group with pain, which included persons with burning and sensory disturbance about the tongue and pharynx. Four of these patients showed salivary disturbance, which may be traced directly to irritation of the chorda tympani and the auriculotemporal nerve; these effects also were relieved. Thirty-five of the forty-two patients with aural symptoms presented varying grades of catarrhal deafness, due either to compression from overclosure or to chronic nasal infection. Four of the patients with aural disturbance presented shortened bone conduction and were not improved by reposition of the jaw; these may be regarded as having either involvement of the eighth nerve or microfracture of a region near the otic capsule which interfered with transmission of sound waves to the inner ear, as observed by Guild. The dizziness is not typical of toxic labyrinthitis. Twenty-six of the series of patients, or about 20 per cent, showed mild herpes of the buccal mucosa, the angles of the mouth and the external auditory canal, which in all cases was preceded by pain and improved with disappearance of the other symptoms. The results of reposition of the jaws were generally good except in a few cases of malocclusion of natural teeth, in which great difficulty was presented. However, it was noted that the conditions showing the best results were corrected in several stages, by slowly increasing the vertical dimension of the jaw. This increase in distance is built into the molar district of the jaw and is not a problem merely of "opening the bite." Some failures to obtain a proper result have been traced to lack of understanding by the dentist of this point and of the anatomic problem involved.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

AN ANALYSIS OF OVER FOUR THOUSAND CASES OF EDUCATIONAL DEAFNESS STUDIED DURING THE PAST TWENTY-FIVE YEARS. MACLEOD YEARSLEY, Brit. J. Child. Dis. **32**:107 (April-June); 196 (July-Sept.); 264 (Oct.-Dec.) 1935.

Hereditary deafness at birth is biologic, while sporadic deafness at birth is a manifestation of disease. A paramount characteristic of hereditary deafness at

birth is that it follows the Mendelian ratio. In the inheritance of deafness hearing appears to be a dominant character and deafness a recessive; hence, in order that the number of persons with hereditary deafness at birth may be reduced, it is important that those known to carry the recessive character should be allowed to mate only with persons who can hear and whose family histories are without taint of deafness. In the present state of society an endeavor to eradicate deafness by preventing the breeding of all persons with undesirable dominant characteristics and of all those with undesirable recessive characteristics would be regarded as grossly unjunst.

Sporadic deafness at birth presents a problem of disease rather than of inheritance. The factors that are involved are those of environment and disease. Better housing, hygiene and ventilation and the reduction of unemployment are a few of the factors.

The subject of acquired deafness and the various diseases that are mainly responsible are discussed in detail.

Finally, the matter of detecting deafness in infants and in persons of preschool and of school age is considered. The series of articles concludes with a discussion of education of the deaf.

LANGMANN, New York. [AM. J. DIS. CHILD.]

ABSCESS OF THE CEREBELLUM. Rev. d'oto-neuro-opht. **13**:727 (Dec.) 1935.

The need of precision and system in making a labyrinthine examination was stressed by Barré, who advocated the recording of the character and tempo of nystagmus, the extent and direction of deviation of each of the extended arms, the use of a plumb line in making the Romberg test and the presence or absence of "vestibular disharmony." The last-mentioned sign may be the first indication of cerebellar disease. Velter said that disturbances of associated movements of the eyes indicate a lesion of the posterior fossa and that deviation of the eyes to the side opposite the lesion is the most characteristic phenomenon of cerebellar abscess.

Halphen reported a case of a cerebellar syndrome complicating chronic sinusitis in which the Bordet-Wassermann reaction of the spinal fluid was strongly positive. The patient recovered promptly with antisyphilitic treatment. Another patient was operated on, and a large cystic arachnoid pocket was emptied; a relapse occurred after a month, and the patient died; autopsy revealed a large cerebellar abscess. De Martel said that many of the serious disturbances in cases of cerebellar abscess arise from obstruction to the circulation of the cerebrospinal fluid; he advised the occipital route as that of choice in operation.

De Morsier discussed the difficulty sometimes encountered in differentiating a pyramidal and a cerebellar syndrome. Cerebral lesions simulating lesions of the cerebellum, according to van Bogaert, are located in the paracentral lobule.

Ody reported the case of a young man with severe headache, vomiting, emaciation, stupor and left hemiparesis, on whom a mastoidectomy on the right side had been performed five months before; healing was apparently perfect. Two weeks before his admission to the hospital, infectious endocarditis had been discovered. A jacksonian convulsion ensued and was followed by complete left hemiplegia, which lasted three days. The following week the same sequence of events was repeated. There were no signs of cerebellar involvement; nystagmus toward the right was noted. Operation on the right temporoparietal region revealed no evidence of abscess. A third crisis supervened, with tonic convulsion, opisthotonos and deviation of the head and eyes to the right. After the seizure there were slowing of the pulse, stiffness of the neck and disturbance of respiration. In spite of the lack of evidence of disease in the mastoid, the old wound was reopened; the dura was exposed, and a subdural abscess was evacuated; the lateral sinus was not thrombosed. Rapid recovery followed. The cerebellar seizure was believed to be due to obstruction of the passages of Luschka and Magendie from edema of the hemisphere or tonsil; compression of the cerebral axis by the swollen organs accounted for the paralysis.

Roger cited the report of Puusepp of 331 cases of abscess of the brain which the reporters failed to mention in their bibliography. In 264 of these cases the abscess was due to injury received in the World War; in 34 it was of otogenic origin; in 1 it followed a pulmonary abscess, and in 1, a fall on the head, without resulting demonstrable fracture of the skull.

Dereux reported the case of a man aged 50 who had a staphylococcic infection causing furunculosis, pulmonary abscess, left occipital osteitis and encephalitis of the cerebellum; three microscopic abscesses, not observed at operation, were seen at autopsy. Dereux maintained that by aid of tests for passivity (hypotonia of André-Thomas) this diffuse inflammation was revealed. Signs of disturbance of tonus often appear earlier than other signs. He questioned the statement that a cerebellar abscess can remain without symptoms until death.

Moriez observed a case of cerebellar abscess associated with psychosis. He attributed the psychosis to associated diffuse encephalitis. Signs of pyramidal involvement were present also and were explained by extension of the softening to the paracentral lobule.

Suñé y Medán reported a case of bilateral chronic otitis, with evident signs of abscess on the right side of the brain. Mastoidectomy on the right side and exploration of the brain revealed no abscess either of the cerebrum or of the cerebellum. Two days later paralysis of the left oculomotor nerve, adiadokokinesis and coma developed. The left mastoid was exenterated, and a large abscess of the cerebellum was evacuated. The patient recovered. Drainage by glass tube and dressings twice a day are advocated in cases of abscess of the brain.

Weill called attention to difficulties in respiration in cases of abscess of the cerebellum. A case in point was that of a man with coma and respiration of the Cheyne-Stokes type. Respiration ceased with the first whiff of ether and again on evacuation of the abscess. Weill described also a case of dementia praecox associated with chronic otorrhea, in which an erroneous diagnosis of abscess of the brain led to a futile operation. He warned that in an adolescent patient with chronic otorrhea accompanied by signs of obtusion, headache and emaciation, early manifestations of dementia praecox should be sought.

Portmann and Despons reported 4 cases of cerebellar abscess observed at autopsy. They expressed the belief that the diagnosis of an encephalic complication can be established only by a grouping of the symptoms noted from day to day. A single sign of meningeal or cerebellovestibular involvement is not sufficient for diagnosis. Cerebellovestibular signs are diffcult to elicit because of the state of the patient or because the tests are not made systematically.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

QUESTION OF ANTERIOR PETROSITIS OR BULLITIS. F. FEDERICI, Arch. ital. di otol. **48**:413 (July) 1936.

Federici reports a case of acute suppuration of the tympanic cavity, with rapid propagation of the suppurative process to the subcochlear and apical cells and the nucha and paralysis of the abducens and trigeminal nerves. An operation was performed, and the suppuration was controlled by treatments through the operative wound. At one of the treatments a fistula on the medial wall of the tympanic cavity to the promontory of the tympanum was discovered and treated. The course ended in recovery. The pathologic process described is known as anterior petrositis. The author discusses the advisability of designating it bullitis, meaning inflammation of the bulla ossea. The name bullitis is indicated because of the location of the suppuration and because of data found in studies of comparative anatomy.

EDITOR'S ABSTRACT.

LABYRINTHINE REFLEXES AFTER THE OTOLITHIC MEMBRANES HAVE BEEN THROWN OFF. T. HASEGAWA, Arch. f. d. ges. Physiol. **236**:589, 1935.

Magnus and de Kleyn found that the postural reflexes were abolished after throwing off the otolithic membranes in guinea-pigs by centrifugation, while the

acceleratory reflexes were still present. Subsequent experiments of Hasegawa, de Kleyn and Versteegh showed, however, that the tonic (postural) reflexes of the labyrinth are preserved after the otolithic membranes have been thrown off. The results of Hasegawa differ from those of de Kleyn and Versteegh in regard to the reflexes on linear acceleration (progression movements). These reflexes were still present in the experiments of de Kleyn and Versteegh on guinea-pigs after violent centrifugation, while they were not noted by Hasegawa after this procedure. Hasegawa repeated the same type of experiments in frogs and corroborated his observations in guinea-pigs. He explains the results of de Kleyn and Versteegh by the assumption that these authors did not succeed completely in throwing off the otolithic membranes.

SPIEGEL, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

INDICATIONS FOR OPERATION IN ACUTE DIFFUSE LABYRINTHITIS IN THE COURSE OF ACUTE SUPPURATING OTITIS MEDIA. J. SPIRA, Monatschr. f. Ohrenh. **70**:695 (June) 1936.

Spira reviews the literature on acute diffuse labyrinthitis and then describes his own observations in eight cases. The first three patients acquired acute diffuse labyrinthitis in the first or the second week of an attack of suppurating otitis media. In these cases conservative measures resulted in complete cure.

The fourth case was that of a patient in whom in the opening of an extradural abscess of the posterior cranial fossa the posterior semicircular canal was probably injured. Within the next few days, acute diffuse labyrinthitis developed. At first, an expectant attitude was taken, but when facial paralysis developed and changes appeared in the cerebrospinal fluid, the operation on the labyrinth was performed and cure resulted.

The fifth case was that of a boy who acquired suppurating otitis media in the course of scarlet fever and diphtheria. In the third week of the otitis media mastoiditis developed, and two weeks later, thrombosis of the sigmoid sinus. Antrotomy was done, the sinus was incised and the thrombus was removed. Several weeks subsequently nystagmus and severe headaches developed. After the protruding wall of a cerebellar abscess was opened, the patient recovered, but after a time it was discovered that the diseased ear was deaf.

In the three last cases the condition developed in the course of an epidemic of influenza. In the first of these cases the labyrinth became involved early—in the stage of serous secretion—and for this reason an expectant attitude was taken, since the literature indicated that in such cases the condition takes a mild course. After two weeks, all symptoms of involvement of the labyrinth had disappeared, but in the fourth week headaches and facial paralysis appeared and spinal puncture revealed pleocytosis. However, surgical intervention did not save the patient. In the next case, the labyrinthitis developed in the second week of an attack of relapsing otitis. After two days fulminating meningitis developed, and death followed. In the last case only mild otitis media developed, which did not result even in perforation of the tympanic membrane. However, a spinal puncture revealed a pleocytosis, and so an operation on the labyrinth was made, which revealed a focus of suppuration. This patient recovered.

In evaluating his experiences the author emphasizes that six of his eight patients recovered and that all were at first treated conservatively, surgical procedures being resorted to only after the examination of the cerebrospinal fluid disclosed involvement of the meninges. An exception to this rule was the case of scarlet fever. The author concludes that in acute diffuse labyrinthitis that develops in acute otitis media, the severity of the infection, the patient's general condition and the condition of the spinal fluid should determine the mode of the intervention. In the course of extremely malignant influenza with sudden exclusion of the function of the internal ear, he recommends a radical intervention, that is, an operation on the labyrinth. For other cases he recommends an expectant attitude, with constant control of the cerebrospinal fluid. Regarding the method

of the operation on the labyrinth, he says that in simple, uncomplicated diffuse labyrinthitis partial operation with wide drainage is sufficient but in labyrinthitis with advanced meningitis a total operation with exposure of the posterior cranial fossa and opening of the internal auditory meatus, is necessary.

EDITOR'S ABSTRACT.

TUBERCULOUS MENINGITIS. JUI-PING WU, Chinese M. J. **50**:506 (April) 1936.

In the past fourteen years 447 cases of meningitis in children were observed at the Peiping Union Medical College, and in 216 of these it was tuberculous. The cases were analyzed as to age of patient, mode of onset, physical findings, type of fever and incidence of common signs. Tables are offered on the presence of tuberculous lesions in other parts of the body, the interval between the detection of beginning tuberculosis and the onset of meningitis. In 50 per cent of the cases the white blood cell count was between 10,000 and 20,000, and the percentage of polymorphonuclear leukocytes was practically always high irrespective of the age of the patient or the duration of the disease. The cell count of the spinal fluid was below 200 per cubic millimeter in half of the cases and was about 500 in approximately 15 per cent, with marked preponderance of lymphocytes at all ages.

In 69 per cent of the cases tubercle bacilli were isolated. In 65 per cent pulmonary lesions were found, the miliary type being more frequent in childhood.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

Pharynx

TREATMENT OF VINCENT'S ANGINA WITH ACETARSONE. C. H. MAXWELL JR., New York State J. Med. **36**:874 (June 1) 1936.

One is advised to use acetarsone locally as a paste or in glycerin. At the same time it should be given internally. Cleansing mouth washes of sodium perborate or hydrogen peroxide were also used in the cases reported. Large amounts of orange juice are advised.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

A CASE OF PHARYNGEAL ASPHYXIA IN A GIRL FOUR YEARS OLD WITH EXOPHTHALMIC GOITER. LOUIS LEROU and E. WELTI, Bull. Soc. de pédiat. de Paris **33**:515 (June) 1935.

A girl 4 years old, who had exophthalmic goiter, acquired acute asphyxia. When a laryngoscope was passed, no abnormality was found beyond the pharynx but the difficulty in breathing subsided. It became evident that the obstruction to respiration was due to huge, inflamed tonsils and falling back of the base of the tongue. The child required the introduction of a Mayo tube into the throat every night for three nights, although she was better during the day. As the tonsillitis subsided, she recovered. The goiter was not large enough to cause obstruction.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

POSTANGINAL SEPTICEMIA. H. WEINHOLD, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:133, 1936.

Weinhold first differentiates two clinical forms of postanginal septicemia—(1) the acute form with abscess and (2) the more chronic form. Further, he gives his attention to the pathologico-anatomic processes in the development of septicemia, taking up first the development by way of the blood stream and then the pathogenesis by way of the lymph passages. After this he describes seven cases. In discussing these cases, he says that in six of them a small abscess was found near the tonsil. There never was an abscess on the cervical lymph nodes, but they were always enlarged and inflamed. The observations revealed clearly that the

infection of the blood stream takes place near the tonsil. The following factors indicate that in the reported cases the general infection originated in the tonsillar veins: (1) the presence of peritonsillar inflammatory processes (nearly always an abscess), (2) the absence of changes in the jugular vein and (3) the efficacy of the surgical exclusion of the palatine tonsils and of their venous discharges.

EDITOR'S ABSTRACT.

DIPHTHERIA AND TONSILLECTOMY. K. A. ROMBACH, Nederl. tijdschr. v. geneesk. **79**:5439 (Nov. 23) 1935.

In view of the successful treatment of a diphtheria carrier, a girl 6 years of age, by adenoidotonsillectomy and the favorable result of this treatment cited in the foreign literature, the author concludes that this method is the most effective with which to cope with the condition. He believes adenoidotonsillectomy to be urgently indicated in the diphtheria carrier and this treatment to be effectual and harmless. VAN CREVELD, Amsterdam, Netherlands. [AM. J. DIS. CHILD.]

Larynx

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES. J. A. COYLE and L. K. SYCAMORE, New England J. Med. **214**:677 (April 2) 1936.

This article contains a historical résumé of the development of instruments for inspection of the larynx, trachea, bronchi and esophagus, with a report of cases, three in children. GENGENBACH, Denver. [AM. J. DIS. CHILD.]

MULTIPLE PAPILLOMA OF THE LARYNX. A. CODINACH Y SEGUGA, Arch. de med. inf. **5**:152 (April-June) 1936.

A case is reported of multiple papilloma of the larynx in a child of 9 years. The condition is rather rare and gives rise to alarming symptoms. It frequently recurs. The best treatment is direct removal and the application of some modifying liquid to the base. SANFORD, Chicago. [AM. J. DIS. CHILD.]

AFTER-TREATMENT OF TRACHEOTOMIZED CHILDREN. E. VON GYÖRGY, Arch. f. Kinderh. **104**:208 (March 29) 1935.

After tracheotomy in a child the following points are important: (1) immunization against diphtheria, (2) prophylactic treatment against pneumonia, (3) keeping the cannula clean at all times, (4) prevention of drying of secretions in the cannula and (5) earliest possible removal of the cannula.

CRANE, Chicago. [FROM THE AUTHOR'S SUMMARY; AM. J. DIS. CHILD.]

Nose

OSTEOMYELITIS OF THE FRONTAL BONE: NOTES ON THREE CASES. H. P. MOSHER, J. A. M. A. **107**:942 (Sept. 19) 1936.

Mosher reports three cases, in two of which there was a fatal outcome. He says that he is convinced even more strongly than when he made his report of three years ago that edema of the skin of the forehead is a rough guide to the extent of infection of the bone and periosteum. If there is actual necrosis of the bone, the bone is infected without necrosis for from 1 inch (2.5 cm.) to 1½ inches (3.8 cm.) beyond the necrotic area. Necrosis of the bone does not occur until from seven to ten days after pitting edema appears, and the roentgenogram is not positive until necrosis appears. Examination of the specimen of bone removed in two of the cases reported showed that the infection spreads along the inner surface of the bone, as well as by the diploic veins. When the infection spreads by way of a diploic vein, it may localize at a point far from the original source of infection. When it does so localize, the pus tends to work both inward and outward,

giving either a subperiosteal abscess or an extradural abscess or both, with destruction of the bone between the two. When a condition has lasted two or three weeks, the operator should expect to find one or both of these conditions. Histologic examination of the author's specimens shows, in addition, that the infection may spread by way of an inner layer of new bone which is formed between the skull and the dura. The small veins which run in the new bone are often infected, and there are numerous hemorrhagic clots which also are infected. Further, the infection spreads by way of the fibrous tissue which covers the new bone and binds the inner surface of the skull to the dura. The operator who is performing his first operation in a case of osteomyelitis of the skull should expect an extradural abscess as a matter of course. In fact, he should expect more than this; he should be on the lookout for subdural abscess or abscess of the brain. The abscess of the brain, if present, is usually seen later or comes later as a complication, but it is always round the corner and should be watched for, even at the first operation. The more Mosher sees of osteomyelitis of the frontal bone, the more he believes that the whole face of the frontal bone should be removed as a routine, from the hair line to the eyebrow. Preferably, it should be removed in one piece. However, if the patient is in poor condition and there is an area of necrosis, it is justifiable to work from the necrotic area outward, removing the bone for from 1 inch to 1½ inches (2.5 to 3.8 cm.) in all directions from the necrotic area. He believes further that both frontal sinuses should be opened and the anterior and posterior walls of each sinus removed. He believes strongly that the lateral limit of the bone flap on each side should be at least the outer angle of each frontal sinus or, better, the outer angular process of the frontal bone on each side. The objection to this extensive removal is the deformity. It has been proved that fully 90 per cent of this can be corrected by modern plastic surgery. Therefore, the surgeon should not allow his hand to be halted by the question of deformity. If he does, he will lose most of his patients with osteomyelitis of the skull.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

RHINORRHEA AND NEOPLASMS OF THE CENTRAL NERVOUS SYSTEM. ROBERT E. BRITT, J. Nerv. & Ment. Dis. **81**:654 (June) 1935.

Spontaneous escape of spinal fluid from the nose is usually associated with severe injury to the head or nose, a nasal infection, a complication of an intranasal surgical procedure, a congenital anomaly, meningitis or a tumor of the brain. The last-named condition is the most frequent cause of drainage of spinal fluid through the nose, and in the two cases reported it was undoubtedly due to increased intracranial pressure.

A 16 year old boy for whom a diagnosis of cerebellar tumor was made presented along with other symptoms and signs leakage of a yellowish fluid from the right nostril. At 4 years of age he suffered from severe frontal headaches, dizziness, unsteadiness of gait and a tendency to fall toward the right. At that time a diagnosis of polioencephalitis was made. Two years later projectile vomiting developed and choked disks and cerebellar signs made their reappearance. Roentgenograms of the skull at that time showed thinning of the bones. Suboccipital decompression was followed by paralysis of the left extremities which lasted four months. Two years later a second operation was performed, and 17 cc. of amber-colored fluid was removed from the cerebellum, as well as tumor tissue. For eight years he received radium treatment at intervals.

Six months prior to death pain developed in the right lower extremity, with numbness in the upper third of the right side of the face, diplopia, convulsions and a discharge of yellowish fluid from the nose.

Lumbar puncture showed xanthochromic fluid under normal pressure. A roentgenogram of the skull showed enlargement of the sella turcica and partial destruction of the posterior clinoid processes.

Stupor and high fever set in twelve days after his admission to the hospital. The patient responded to medication at first but died five weeks later.

Postmortem examination showed a tumor in the posterior fossa. There were thinning of the bone of the skull, erosions of the lamina cribrosa and tears in the pia-arachnoid over the region of the right orbital and subcallosal and both temporal convolutions. "The cortical tissues in these regions contained lacerations which permitted the escape of cerebrospinal fluid through the lamina cribrosa." Partial destruction of the posterior clinoid processes as well as enlargement of the sella turcica can be accounted for on the basis of distention of the pia-arachnoid from accumulation of spinal fluid in the floor of the third ventricle (internal hydrocephalus).

The second case was that of a man 28 years of age, who was apparently suffering from a deep-seated neoplasm in the brain. His symptoms of tumor appeared first in 1921. Rhinorrhea set in in 1931. Britt attributes the nasal discharge of spinal fluid together with other neurologic findings which the patient presented to erosion of the sella turcica or internal hydrocephalus.

JAHR, Omaha. [AM. J. DIS. CHILD.]

THE "RUNNING" NOSE: CLINICAL RECOLLECTIONS AND REFLECTIONS. G. EWART MARTIN, Edinburgh M. J. **43**:498 (Aug.) 1936.

Martin is a surgeon in the department for diseases of the ear, nose and throat of the Royal Infirmary, Edinburgh. He states that as otitis media comes by way of the nasopharynx and nose, there would be much less infection of the ear and, generally, much less deafness if more attention were paid to the nose, which as a focus of infection is as important as the pharynx or the tonsils. A child with a running nose goes everywhere; a child with sore throat is kept at home.

The author discusses the anatomy and physiology of the nose and the moistening of the inspired air by the normal constant secretion which in an adult amounts to 17 ounces (503 cc.) per day, depending on the dryness or the moistness of the atmosphere. Normally one side of the nose is more active than the other. Whenever there is blocking of one side from disease or some other abnormality, the secretion which is formed on that side is not picked up by normal air, and it falls down to the floor of the nose, coagulates, drops backward or slips forward and gives the first indication of a running nose.

In persons with adenoids both airways become blocked, and this produces mouth breathing during the day or night. The chest cannot be sufficiently expanded because of the lack of moisture.

Martin discusses the disturbed filtration of inspired air caused by the failure of the nose to continue as a filter for germs when the mucous membrane is disturbed. Foreign matter in the nose arrests the filtering action of the cilia.

How effective is the use of drugs in the nose? Those drugs which clog the mucous covering interfere with the activity of the cilia. In a normal healthy person the majority of pathogenic organisms entering the nose are destroyed. The nasal secretion must be preserved. The only douche should be one of an isotonic solution, the only oil used a volatile one. The author objects to continuous treatment, especially with one type of medicine, because of interference with ciliary action. In early inflammatory conditions the prevention of blocking may be possible by the temporary and scanty use of drugs which shrink the nasal mucosa.

The author then discusses the treatment of common colds and the question of the removal of adenoids and tonsils.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

RADICAL THERAPY OF SUPPURATION OF THE FRONTAL SINUSES. M. HAJEK, Monatschr. f. Ohrenh. **70**:641 (June) 1936.

Hajek proves that it is inadvisable to adhere to one certain method in the treatment of suppuration of the frontal sinuses and recommends that the treatment should be adapted to the individual case. The endonasal conservative method produces satisfactory results in the majority of cases of acute or chronic empyema of the frontal sinuses in which the bony frame is not involved. If the endonasal method does not produce the desired results, simple trepanation is indicated, but

only if the bone is completely intact and there is no old empyema. A cure through trepanation alone can take place only if the endonasal excretory duct can be exposed. If this is impossible, an artificial duct must be made. The removal of the diseased mucous membrane of the frontal sinus has to be dispensed with in these cases. In acute empyema with threatening or accomplished perforation into the orbit, the orbital roof should be exposed, and after emptying of the orbital abscess and removal of the necrotic portions of the bone, it is advisable to make a wide opening and insure a functioning duct. The mucous membrane and the other parts of the bony framework should remain intact. If similar complications arise in case of chronic empyema of the frontal sinus, the same measures are advisable as are recommended for acute empyema. However, if the bone has become extensively involved, not only the orbital plate but the anterior wall and the mucous membrane has to be removed. Depending on the condition of the frontal sinus and on the extent of involvement of the bone, the method of Kuhnt, Killian or Riedel can be employed and can be followed later by cosmetic operations.

EDITOR'S ABSTRACT.

EXPLORATORY ENDONASAL TREPANATION OF THE LOWER WALL OF THE FRONTAL SINUS. E. I. MATIS, Monatschr. f. Ohrenh. **70**:656 (June) 1936.

Matis shows that in the diagnosis of disorders of the frontal sinus the customary methods of examination, such as endonasal examination, roentgenoscopy, exploration and irrigation of the frontal sinus, are frequently inadequate. For all cases in which the diagnosis proves difficult, he recommends his method of endonasal exploratory trepanation of the frontal sinus. Using local anesthesia, he makes an arc-shaped incision at the edge of the apertura pyriformis. Then the soft parts are pushed aside until the lower wall of the frontal sinus is completely exposed, an opening is made above the frontomaxillary suture and the cavity of the frontal sinus is inspected.

EDITOR'S ABSTRACT.

Miscellaneous

MENINGITIS DUE TO BRUCELLA IN A CHILD. MARY A. POSTON and ROBERT H. THOMASON, Am. J. Dis. Child. **52**:904 (Oct.) 1936.

Poston and Thomason report the case of a girl with low grade, subacute meningitis due to Brucella abortus, who recovered completely after repeated spinal punctures and intraspinal injections of human immune serum. It is possible that conditions diagnosed as tuberculous meningitis in which no organisms were seen on direct smear and some in which "influenza-like" bacilli were seen but not cultured may have been meningitis due to Brucella.

WAGGONER, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

THE PROBLEM OF THE ASTHMATIC CHILD. FREDERICK W. SCHULTZ, Illinois M. J. **69**:33 (Jan.) 1936.

In a review of the accepted conception of juvenile asthma Schlutz states that from the standpoint of symptoms three principal forms, or types, of asthma may be recognized in childhood: asthmatic bronchitis, characteristic of the period of infancy and observed frequently after the second year; true asthmatic attacks beyond that period and up to puberty, resembling in every symptom the adult type, and asthmatic cough, observed generally beyond infancy and characterized by a specific sound, extraordinary duration, resistance to medication and remarkable freedom of the lungs from any inflammatory change.

A striking feature of nearly all forms of asthma in childhood is their peculiar relationship to the manifestations of exudative diathesis and to lability of the nervous system. This is the constitutional phase which colors the background of practically every asthmatic person. All evidence indicates that asthma is practically always an allergic reaction.

The most important predisposing cause of asthma and of practically all allergic conditions is heredity. Food becomes relatively unimportant as a factor in sensitivity after the fifth year. Then the air-borne substances are in the ascendant and allergens are derived from focal infections. In asthma due to infection, the causative lesion is invariably located in the upper part of the respiratory tract.

Protein sensitization alone does not entirely explain all the manifestations associated with allergy. The theory has been advanced that the basis of allergy is a disturbed balance between the sympathetic and the parasympathetic nervous system. When the physicochemical balance is profoundly disturbed, various nonspecific and specific factors acting together will produce asthma and precipitate the attack.

The striking characteristics of the asthmatic state in childhood are persistent chronic cough and dyspnea during the actual attacks. Eosinophilia may be present and marked or may not be present. The pathologic picture in the lungs is definitely noninflammatory.

The treatment of asthma is a complex problem and involves a number of factors. Schultz expresses the belief that it is of prime importance to discover the specific allergen or allergens which may be etiologic factors in a given case and if possible to have them removed from the environment. He believes that if this is difficult or if such important foods as milk and eggs are involved, the gradual desensitization can be brought about by the injection of specific allergens.

Asthma precipitated by allergens originating in focal infections is probably the most common type the practitioner will encounter. An early and thorough tonsillectomy and adenoidectomy will, he thinks, bring a great deal of relief. When infection of the sinuses is the focus, the situation is more difficult. Autogenous vaccine may be of some value.

For the acute attack Schultz prefers epinephrine, ephedrine, or perhaps atropine, and as a last resort change of climate.

BARBOUR, Peoria, Ill. [AM. J. DIS. CHILD.]

EVALUATION OF MERCURIAL ANTISEPTICS IN THE PRESENCE OF SERUM. DOROTHEA E. SMITH, E. J. CZARNETZKY and STUART MUDD, J. Bact. **31**:7 (Jan.) 1936.

Smith, Czarnetzky and Mudd compare the germicidal action of several mercurial antiseptics in a solution of sodium chloride and in 80 per cent serum. An accurate method of plating was used, and the time and concentration required for killing 50 per cent of a culture of a smooth strain of Bacterium typhosum was taken as a basis of comparison. Metaphen, merthiolate, mercurochrome and mercury bichloride all combine with the proteins of the serum. The ratio of the rate with which the mercury of the mercurial compounds combines with bacteria to the rate with which it combines with the proteins of the serum is more favorable in the case of some of the mercurial compounds than with the others. The authors express the belief that these compounds kill bacteria only in proportion to their content of mercury. The decrease in germicidal activity of the compound when it is used in serum is proportional to the amount of mercury chemically combined with the serum. Thus, it seems that the effect of serum is such that metaphen, merthiolate, mercurochrome or mercury bichloride may not be administered intravenously as an antiseptic.

STOESSER, Minneapolis. [AM. J. DIS. CHILD.]

OCCURRENCE OF TRIGEMINAL NEURALGIA IN PATIENTS HAVING MULTIPLE SCLEROSIS. J. M. MEREDITH and GILBERT HORRAX, J. Nerv. & Ment. Dis. **82**:497 (Nov.) 1935.

Pain in cases of multiple sclerosis is rare. Other sensory disturbances, such as numbness and paresthesia, are comparatively common and probably occur at some period in the course of the disease in the majority of patients. The pathogenicity of pain in the face in cases of multiple sclerosis is unknown. The theory that it may be due to plaques of sclerosis in the pons, the medulla or the descend-

ing spinal root of the fifth cranial nerve is unlikely, in view of the relief obtained by injections of alcohol or avulsion of the sensory root peripheral to these lesions. It is further refuted by the reported cases of multiple sclerosis in which lesions similar in type and location were discovered and no pain in the face was noted in the history.

Twenty-five instances of concomitant trigeminal neuralgia and multiple sclerosis were encountered in a review of cases reported in the last seventeen years. In some of the patients pain preceded the symptoms of multiple sclerosis, whereas in others the reverse was true. It is probable that trigeminal neuralgia complicated by multiple sclerosis is more likely to be bilateral than the ordinary form of the disease.

Two additional cases are reported in some detail, in one of which bilateral trigeminal neuralgia occurred at different periods. Both patients were operated on, with subsequent complete relief from pain for one year. Inhalations of trichlorethylene and injections of alcohol were effective in relieving the pain temporarily in the two cases. Modern methods of treating trigeminal neuralgia associated with multiple sclerosis are as effective as in ordinary forms of the disease.

HART, New York. [ARCH. NEUROL. & PSYCHIAT.]

SUGAR IN THE BLOOD AND THE SPINAL FLUID. HEINZ BOETERS, Ztschr. f. d. ges. Neurol. u. Psychiat. **154**:462 (Jan.) 1936.

The Hagedorn-Jensen method of quantitative estimation of sugar in the spinal fluid is the most reliable. The spinal fluid should be examined as soon as possible after it is drawn. Readings for the blood and the spinal fluid should be taken at the same time. Boeters reports 986 simultaneous determinations of the sugar content of the blood and the spinal fluid. This material is large enough to be statistically reliable. The reliability of the Hagedorn-Jensen method was shown by study of the difference of two separate quantitative determinations of sugar in the same specimens of spinal fluid and of blood. The average difference in 926 such double determinations of blood was 3 mg., and in 761 studies of the spinal fluid, 1 mg.

A so-called "normal" series of 285 cases were studied. They represented various functional diseases of the nervous system. The average reading for the blood sugar was 98.7 mg. per hundred cubic centimeters, ranging frorm 75 to 125 mg. The average reading for the sugar content of the spinal fluid was 63.2 mg., ranging from 45 to 75 mg. These results do not agree with those of some investigators, who found a wider variation in the sugar content of normal spinal fluid. The ratio of the sugar in the spinal fluid to that in the blood is about 2:3, the sugar content of the spinal fluid in this series being 65.15 per cent that of the blood.

Two hundred and twelve cases of dementia paralytica were studied. The average amount of sugar in the spinal fluid was 53.4 mg., as compared with normal figures for the blood sugar. The median of the ratio of the sugar of the spinal fluid to that of the blood was 55, as compared with the median for the "normal" series, of 63.2 per cent. The sugar level of the spinal fluid is lower in the earlier stages, in which the patients present themselves for the first time for treatment—50.1 mg. in 45 cases. An absolute and relative diminution of the amount of sugar in the spinal fluid in dementia paralytica was noted in cases in which pleocytosis was more marked (49 mg., in 52 cases in which the cells were over 30, and 54 mg., in 95 cases in which the cells were below 30). In 61 cases of tabes dorsalis and cerebrospinal syphilis, the average reading for the sugar in the spinal fluid was 59 mg., being 58.7 per cent of the average reading for the blood sugar. In 2 cases of acute multiple sclerosis the reading was 37 and 45 mg., being 45 and 51 per cent, respectively, of the value for blood sugar. In 13 other cases of disseminated sclerosis there were normal readings. In 3 cases of tumor of the brain with reactive pleocytosis, the readings were similarly relatively low.

In 56 cases of meningitis the readings for the spinal fluid varied from 12 to 62 mg., the median being 37.7 ± 4.5 mg.; in 49 cases the median value for the spinal fluid was 36.6 per cent of that for the blood, ranging from 13 to 65 per cent. In these instances also there was a direct relation between the number of cells and the amount of diminution of sugar in the spinal fluid. No difference was noted between tuberculous and other bacterial meningitides. Alimentary hyperglycemia in this series resulted in a corresponding increase in the sugar of the spinal fluid, with preservation of the usual ratio. In 17 cases of hydrocephalus and increased intracranial pressure there was a constant increase in the sugar of the spinal fluid—76 mg. (ranging from 72 to 89 mg.), with normal values for the blood sugar. In 7 cases of tumor of the brain, in which the intracranial pressure was increased, there were similar results (from 70 to 83 mg.). The slight increase in the sugar of the spinal fluid in epilepsy, injuries to the head, chronic encephalitis and cerebral arteriosclerosis is of questional significance and is not statistically reliable. Five double determinations in 2 cases of schizophrenia, with death in catatonic excitement, showed constant hyperglycorrhachia (from 77 to 88 mg.). Other investigators have reported a constant increase in the sugar of the spinal fluid in acute encephalitis. No such cases were available in the course of this study. Significant alterations in the sugar of the spinal fluid were not found in various intoxications with industrial poisons (phosphorus, carbon monoxide, magnesium and benzine), in 2 cases of pellagra, in the myopathies, in Friedreich's ataxia, in idiopathic paralysis agitans and in Pick's and Alzheimer's disease. The only practical clinical application of such studies on the sugar content of the spinal fluid is in cases of bacterial meningitis. The tendency to hypoglycorrhachia does not have much clinical value, though it may be used as an indicator that organic disease is probably present.

SAVITSKY, New York. [ARCH. NEUROL. & PSYCHIAT.]

TUBERCULOUS MENINGITIS WITH RECOVERY. Y. V. SOLOVEY and I. S. ROZENSVEYG, Vrach. delo **18**:381, 1935.

Solovey and Rozensveryg report two cases of tuberculous meningitis with recovery under high voltage roentgen therapy to the spine.

One patient was 33 years of age. The symptoms were typical of meningitis, and the specific organism was found in the cerebrospinal fluid on two different occasions. The treatment was started fifteen days after the onset of the illness, and the patient received five courses of high voltage roentgen therapy to the head and spine. He was discharged in good condition three weeks after the beginning of treatment.

The second case was in a child 2½ years of age. The clinical symptoms, though mild, were characteristic of the disease, but the specific organism was found in the cerebrospinal fluid only on one occasion and in very small numbers. The treatment was started twenty days after the onset of the disease. The child slowly improved and five months later was found entirely normal.

The authors believe that the recovery in these cases was not a temporary remission, as in many of the supposedly recovered patients with tuberculous meningitis, as could be judged by the fact that no recurrences were observed in the two patients in the course of one and a half years of observation.

BODER, Los Angeles. [AM. J. DIS. CHILD.]

INVESTIGATION OF RESORPTION OF SUBSTANCES THROUGH CUTANEOUS AND MUCOUS SURFACES: AN ATTEMPT TO IMMUNIZE BY PERCUTANEOUS APPLICATION OF VACCINE (EXPERIMENT WITH TYPHOID VACCINE). S. MATSUMOTO, Acta dermat. **25**:92 (June) 1935.

Percutaneous application of 1 Gm. of ointment containing 13 mg. of typhoid vaccine to the skin of the rabbit caused a marked increase in the agglutination power of the blood. A high agglutination titer could also be produced by daily

percutaneous application of from 5 to 10 mg. of typhoid vaccine during two or three weeks on an area of several square centimeters of skin. If a rapid rise of immunity is desired, the dose of vaccine may be safely increased without fear of any considerable local or general reaction. Vaccine ointment containing 100 mg. of typhoid bacilli when applied to the skin and replaced three times a week creates immunity of a satisfactory strength without producing any by-effects.

Matsumoto has found that the percutaneous application of vaccine is a convenient means of bringing about active immunization without producing any reaction, although an enormous quantity of bacteria is used as compared to the dosage given by the subcutaneous or intravenous method. The skin acts as a filter against invasion of the substance in the vaccine which usually produces the by-effects.

The need of a larger quantity of vaccine for such a method will probably make its use on a larger scale a little difficult, but, on the other hand, when the vaccine is used percutaneously it is unnecessary to purify it for the purpose of diminishing untoward reactions.

BLOOM, New York. [ARCH. DERMAT. & SYPH.]

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

Seventieth Annual Meeting, Long Beach, N. Y., May 27-28, 1937

EDMUND PRINCE FOWLER, M.D., *President, Presiding*

ABSTRACTED BY DR. ALFRED LEWY, CHICAGO, FOR DR. ROBERT SONNENSCHEIN

Thursday Morning Session, May 27

SYMPOSIUM ON THE NEURAL MECHANISM OF HEARING

I. ANATOMY AND PHYSIOLOGY

PERIPHERAL APPARATUS (IN THE TEMPORAL BONE). STACY R. GOULD, PH.D., Baltimore.

In view of the limited time available, no attempt is made to review the enormous literature on theories of hearing or to present a systematic description of the anatomy of the inner ear. Instead, emphasis is placed on the fact that it is not yet known how sound waves cause nerve impulses to be set up in the fibers of the cochlear nerve. Attention is directed to certain details of the structure of the organ of Corti that are omitted from most textbooks—in particular, to the relations of the outer hair cells to other structures. No theory of hearing is propounded or defended, but the suggestion is made that writers of theoretical papers on the physiology of hearing have not taken sufficiently into account the possibilities presented by movements within the organ of Corti during the passage of sound waves through the fluids of the cochlea. It is also pointed out that the biochemistry of the inner ear is almost completely unknown.

CENTRAL PATHWAYS. DR. R. LORENTE DE NÓ, New York.

There are several points in the papers presented that I should like to discuss. One relates to the remarks of Dr. Wever about the localization of tone. I shall endeavor to make clear that "localization of tone" does not mean, at least to my mind, that every tone stimulates only a discrete part of the cochlea, without producing any effect in the neighboring zones of the basilar membrane. On the contrary, the fundamental concept in the theory of localization is that in the auditory system there is a possibility for the nerve impulses to be accompanied by a local sign from the cochlea up to the cerebral cortex. No matter how much the tones may spread in the cochlea, since each tone activates a certain number of the same hair cells that are activated by other tones, the local sign will be carried up to the cerebral cortex, because there are in the auditory system pathways which establish an anatomic projection of the cochlea.

A similar situation holds with respect to the optic system. In that system there is a physiologic point to point projection but not a perfect point to point anatomic projection. In short, localization in the auditory as well as in the optic system means that, because of the anatomic arrangement of the peripheral and central pathways, it is possible for the cerebral cortex to "recognize" at which part of the retina or of the cochlea the activity (or the greater part of the activity) is taking place.

Attention should be called to two recent papers by Hallpike, Hartline and Smith. In relation to the electrical response of the cochlea, these authors have come to conclusions similar to those of Dr. Bast. They have tried to determine

whether the electrical response of the cochlea might be the mechanism of stimulation of the nerve fibers, but they found that apparently this is not the case, because the action potentials of the responses of the fibers of the eighth nerve do not follow the changes that may be produced in the electrical response.

Dr. Hughson's slides show the presence of a number of degenerated fibers around the olive. This is all that one may expect to see in Marchi preparations. As described by Ramón y Cajal, the endings within the olive are unmyelinated; consequently, by means of degeneration methods one can demonstrate only the parent fibers before they divide and lose their myelin sheaths. Dr. Hughson's results rather confirm the classic descriptions of the anatomic connections of the superior olive.

POINT OF VIEW OF THE PHYSICIST. HARVEY FLETCHER, PH.D., New York.

The physicist contents himself with making measurements external to the ear. From such measurements he infers something about the mechanism of hearing. Measurements of the intensity of sound produced at the threshold of hearing enable him to calculate the sensitivity of the nerve endings. It turned out that this sensitivity is as great as it possibly could be without perception of the jostling of the molecules due to the thermal agitation. Measurements of the minimum perceptible differences in pitch and also measurements of the masking effect of thermal noise give data which when properly interpreted enable the physicist to calculate the position on the basilar membrane of the maximum stimulation for tones of different frequency. By making measurements of the loudness of tones and its relation to the masking effect, one can determine approximately the relation between the number of nerve discharges per second and the stimulation expressed in terms of the power necessary to excite the threshold.

II. PATHOLOGY

ENDOLYMPH AND PERILYMPH SYSTEMS. BARRY JOSEPH ANSON, PH.D., Chicago.

In otosclerosis transmission of sound is interrupted by fixation of the last ossicle in the chain (the stapes) to the margin of the orifice in which it rests; the condition is somewhat comparable to that of "stiff joint" at the knee, elbow or hip. The tissue which thus presses on the stapes is a product of the bony capsule in which the organ of hearing is housed and, almost invariably, of a special part of the capsule, through which passes a minute fissure. In the immediate neighborhood of the fissure is an assemblage of tissues more like those of the embryo than is encountered elsewhere in the adult human skeleton. One of these is simple cartilage, which, it is believed, gradually fills the fissure, as if the latter were an unnatural cleft which the organism would obliterate. Once formed, bone of a primitive sort invades the cartilage, but the process does not terminate abruptly; spread of bone continues, as it might in a tumor of slow growth, replaces the normal tissue of the recess in which the stapes is lodged, impinges on the ossicle itself and fastens it immovably.

From a large number of ear bones my associates and I are studying this area minutely, in the hope that by learning exactly what the nature of the successive tissues is we may understand the underlying causes of otosclerosis. A paper will report the progress of this investigation, which I am conducting with Drs. J. Gordon Wilson and T. H. Bast.

THE COCHLEAR NERVE, SPIRAL GANGLION, CENTRAL PATHWAYS AND CORTEX. DR. F. H. LEWY, Philadelphia.

Some highlights of the pathology of nerve deafness are discussed so far as they contribute to better understanding, localization treatment and prevention of nerve deafness. The frequency of birth injuries involving the acoustic nerve is mentioned, and the attention of the obstetricians is directed to the importance of this possibility, which represents a condition similar to that responsible for

spastic infantile paralysis. Histologic examination of material from tabetic patients with progressive deafness indicates that the decrease in hearing often is the consequence not of an untreatable tabetic degeneration of the auditory nerve but of an active syphilitic inflammation which can still be treated successfully. . . ." Similar conditions are found in deafness following measles, scarlet fever, typhoid and influenza. They show that this form of deafness is much more frequently the result of inflammation of the brain and its membranes in the neighborhood of the auditory nerve than of neuritis of the nerve itself. Both these experiences suggest the importance of early examination of the spinal fluid for signs of inflammatory processes and the importance of subsequent therapeutic drainage. Another group of patients suffering from nerve deafness on which pathologic examination throws a new light are those with tumor of the cerebellopontile angle. Clinical and pathologic differentiation of the tumors located within the central nervous system or originating in its membranes from those of the various parts of the acoustic nerve itself may assist in an earlier diagnosis and a more effective surgical approach.

PATHOLOGY OF THE ORGAN OF CORTI. DR. MOSES H. LURIE, Boston.

Correlation of pathologic changes in the organ of Corti with the loss of hearing of animals gives definite clues as to requisites for the prediction of functional loss from examination of histologic sections.

Changes present in the stria vascularis and the cells of Hensen and of Claudius give no information as to the amount of loss of function. The loss could be determined only when there were definite changes in the hair cells of the organ of Corti. The external hair cells degenerated or showed pathologic changes before the internal hair cells did. With the internal hair cells present and the external hair cells degenerated, there was a partial loss of function. With both the internal and the external hair cells degenerated, there was a complete loss of function.

As a result, the emphasis in examination of the organ of Corti has been on the presence or absence of the hair cells only. If the nuclei of the internal and external hair cells can be made out, normal function is assumed for these cells, and in their absence, loss of function. On this basis definite predictions have been made as to the loss of function, which have been found to approximate closely the electrical audiogram of the cochlea.

Various animals deaf from operation on the cochlea, long exposure to intense tones or chemical poisoning and also congenitally deaf animals have been studied from this point of view. With all these types of deafness the prediction of loss of function has been found to agree with the electrical audiogram.

In the congenitally deaf waltzing guinea-pig, the cochlea of which gave no electrical response, no hair cells were present in the organ of Corti, though the other structures, such as Reissner's membrane, the stria vascularis and the tectorial membrane, appeared normal. Some of these animals showed an interesting transition from absence of the organ of Corti in the basal whorls to an almost normal organ of Corti in the helicotrema. In these animals, a response was obtained up to 500 cycles per second at high intensities.

Several animals tested electrically showed losses of from 30 to 40 db. throughout the range of hearing. Histologic examination showed degeneration of the external hair cells of the organ of Corti. The cause of this degeneration was not known and was probably secondary to some disease.

A number of guinea-pigs made partially deaf by exposure to intense tones for long periods also showed definite degeneration of the external hair cells and of occasional internal hair cells. These animals showed loss of function, according to the electrical audiogram of from 20 to 60 db., depending on the amount of degeneration present.

Cats (including Dr. Dworkin's) operated on to produce deafness showed similar changes. The predictions made from the histologic examination checked well with the electrical audiograms.

Hemorrhage into the scala may cause deafness by physical interference, but the amount of functional loss could not be estimated from my examinations. Collapse

of Reissner's membrane with a normal organ of Corti present also presented difficulties in the determination of functional loss, as this also appears to be physical interference. I have found that with this condition present the electrical test gives harmonics for the region involved, as shown by marked distortion of wave forms in the cathode ray oscillograph.

The pathologic changes in the spiral ganglion and the cochlear nerve have not been mentioned because experiments on the correlation of the animal's loss of hearing with the changes in the spiral ganglion, cochlear nerve and organ of Corti are now being carried out.

Estimation of the amount of functional loss of the organ of Corti can be based only on study of the hair cells. This loss should be estimated on the basis of the presence or absence of the external hair cells or of the presence or absence of both external and internal hair cells. I have not found external hair cells present and internal hair cells absent in any of the animals examined.

With loss of the external hair cells, a functional loss of from 30 to 40 db. can be estimated. With absence of both external and internal hair cells, the loss may be estimated as from 60 to 90 db., depending on the extent of the lesion. The larger the lesion, the greater the intensity of the sound needed to spread over the gap. It is therefore important that in testing animals one use stimulation at the threshold rather than maximum stimulation. Maximum stimulation will cause any normal or partially normal organ of Corti to respond, giving an end-result that cannot be interpreted in terms of the pathologic involvement found in the organ of Corti.

III. ANIMAL INVESTIGATIONS

Behavioral, Electrical and Anatomic Studies of Abnormal Ears. Dr. Moses H. Lurie, Boston; Dr. Hallowell Davis, Boston, and Dr. Simon Dworkin, Montreal, Canada.

Cats were trained to open a food box when a pure tone was sounded. The sensitivity of hearing was thus determined for various tones. The inner ears were partially destroyed by operation, and the hearing was retested over a period of weeks. The electrical activity of the cochlea was then measured, and finally the inner ears were studied microscopically. When allowance is made for the limitations of both methods of testing, there is a fair degree of correspondence between the results of behavioral and those of electrical tests and also between the results of such tests and the extent of anatomic damage observed microscopically. True hearing and also electrical activity depend on intactness of hair cells of the organ of Corti on the basilar membrane. Electrical activity in response to a restricted range of loud tones was associated with the persistence of an isolated patch of intact hair cells; conversely, an ear which electrically showed a well defined "tonal gap" was found to have a zone of local damage to the organ of Corti. The locations so determined for the reception of particular tones agree accurately with those previously observed in experiments on guinea-pigs and inferred from psychologic measurements on man.

The saccule was anatomically normal in several ears which were functionally deaf and electrically inactive. This portion of the inner ear does not belong to the auditory system.

Mapping the Cochlea, the Acoustic Pathways of the Medial Geniculate Body and the Auditory Cortex. E. A. Culler, Ph.D., Urbana, Ill.

The program at the University of Illinois Animal Hearing Laboratory consists in mapping the cochlea, the acoustic pathways of the medial geniculate bodies and the auditory cortex.

Two years ago a map of the cochlea of the guinea-pig was published from this laboratory, based on a careful survey of cochlear potentials generated by air-borne frequencies ranging from 60 to 7,000 cycles. This survey differed from all similar efforts in that the cochlea, instead of being drugged, punctured or cauterized, was normal in every respect. The map revealed that each audible

frequency has its own focus of response within the cochlea and that the site of a given focal point can be fixed with a standard error of 5 per cent. To be wholly conclusive, any such chart requires corroboration by tests of hearing after localized lesions. Normal limens are first measured in the guinea-pig after destruction of one ear. The cochlear wall is then punctured with a surgical needle. A tiny perforation is left, but it soon heals over.

Figures to be presented at the meeting indicate that the maximal loss of hearing always occurs at or near the charted frequency; the losses, however, extend over a wide range of frequencies. The following general conclusions may be safely inferred.

(*a*) The resonant principle, being now validated by the concurrent testimony of both cochlear response and hearing tests, seems to be proved beyond any reasonable doubt.

(*b*) These tests provide the first satisfactory evidence of direct agreement between cochlear response and hearing.

(*c*) They indicate that the map of cochlear responses previously published is correct.

The medial geniculate body being the principal way station on the route from the ear to the cortex, its importance for hearing need not be argued. Auditory limens for pure tones are obtained in cats by the conditioned response technic for the seven octaves from 125 to 8,000 cycles. By means of the Horsley-Clarke stereotaxic instrument, localized electrolytic lesions are made bilaterally (symmetrically) in the medial geniculate bodies. After recovery from the operation the cats are again tested on the same frequencies to determine the nature and extent of the loss of hearing.

Results to date permit these two generalizations: (*a*) Complete destruction of one medial geniculate body causes a general loss at all frequencies comparable to the loss from destruction of one hemicortex or one cochlea, and (*b*) localized bilateral lesions affect certain frequencies and not others, proving that the several frequencies traverse the geniculate bodies by discrete pathways. In other words, the localization which begins in the cochlea is maintained through the medial geniculate bodies.

Work on the auditory cortex, while well under way, does not yet permit any assured generalizations.

EXPERIMENTS ON THE MECHANISM OF ACOUSTIC IRRITATION IN THE COCHLEA. DR. HEINRICH G. KOBRAK, Chicago.

The physiologic stimulus of the cochlea consists in dynamic changes in air pressure which are transformed into vibrations of the labyrinthine fluid. For the purpose of analysis, changes in static pressure were studied first. The displacement of the intralabyrinthine fluid due to changes in static pressure in the outer acoustic canal and in the cavity of the middle ear was measured. The curves demonstrate that a change of pressure is far more effective when applied in the middle ear than when applied in the outer acoustic meatus. From the quantitative findings, one must assume that minor changes of pressure in the middle ear, such as those due to swallowing, sneezing or yawning, are effective on the inner ear. Functional tests in the rabbit reveal that the function of the cochlea is not impaired by these minor changes of pressure.

Identical experiments were carried out with changes in dynamic (acoustic) pressure. Sound was conducted by tubes into both the outer and the middle ear. The hearing of the animal was determined by the acoustic reflex of the muscles of the middle ear. The acoustic irritation of the cochlea is definitely better when the sound is applied into the middle ear.

The phenomenon was also studied under pathologic conditions. One coil of the cochlea was destroyed in rabbits. As a result of the cochlear lesion, degeneration of nerve fibers develops in the primary acoustic centers. As is to be expected, these animals with experimental nerve deafness show a substantial loss of function for sound applied into the outer acoustic canal. However, by appli-

cation of sound into the cavity of the middle ear a better hearing function can be demonstrated.

The experiments demonstrate that the cochlea is stimulated both by sound entering through the oval window and reaching the nerve endings through the scala vestibuli and by sound waves passing through the round window and the scala tympani.

ORIGIN OF COCHLEAR POTENTIALS. T. H. BAST, PH.D., Madison, Wis.

In a series of observations on guinea-pigs, it was found that in this animal the development of a normal cochlear potential in response to air-conducted or bone-conducted tones does not depend on the organ of Corti, on the tectorial or Reissner membrane or on an intact cochlear duct. The results also offer strong evidence against the theory of origin of these potentials in the nervous system. Passage of a strong polarizing current through the cochlea has no effect until evident electrolysis occurs, producing, as shown by subsequent histologic examination, extensive disruption of membranes and gross physical damage. Various toxic materials applied to the membrane of the round window produce little or no effect except through damage to the middle ear. The same is true if these materials are introduced directly into the cochlea, provided no serious physical damage is done to this structure and effects on the middle ear are avoided.

The cochlear response appears to be extraordinarily resistant to all influences not causing gross physical change. It has been previously shown that disturbance of pressure relations between the endolymph and the perilymph system will greatly reduce or abolish the response. This observation, together with those reported at this time, led me to the conclusion that the cochlear potentials have a purely physical origin. In a vibrating mechanism consisting of aqueous phases separated by membranes, such as the cochlea presents, it would appear that electrical potentials inevitably result from the physical pattern present.

AN EXPERIMENTAL STUDY OF THE NEURAL MECHANISM OF HEARING. DR. WALTER HUGHSON, Abington, Pa.

In a large series of experiments, sharply localized lesions of the auditory tract have been produced while an intact blood supply to the ear was maintained. After a sufficiently long interval for degeneration to take place the animals were tested by the electrical method. Both acoustic and oscillographic measurements of the response of the cochleas and the nerves were made, and the temporal bones and the brain stem were prepared for histologic examination. Regardless of the extent of atrophy of the nerves or ganglion cells (in the case of the spiral ganglion actual cell counts have been made), the cochlear response is unaffected in regard to frequency and intensity. True action currents, however, cannot be obtained from such a preparation, and the spread effect is also sharply reduced or eliminated.

In material from the brain stem prepared with the Marchi stain, the degeneration of the auditory tract has been studied. Specific lesions of the dorsal and ventral cochlear nuclei and the tuberculum acousticum have made possible a careful analysis of the contribution each makes to the more central pathways. From the latter study it is apparent that the accepted course of the auditory tract in the cat must now be modified somewhat. In none of this material was either the olive or the para-olive included in the degeneration.

IV. EXPERIMENTAL, CLINICAL AND DIAGNOSTIC METHODS

DIAGNOSIS OF NERVE DEAFNESS. DR. SAMUEL J. CROWE, Baltimore.

It is emphasized that the real advances in clinical otology always have and always will come from the man who treats sick patients and not from the laboratory. Experimental work is most valuable, but its chief function is to suggest

new facts and ideas to the practicing otologist. The otologist cannot do his part in applying these ideas and advancing the knowledge of his profession unless he has a broad training in the embryology, anatomy, physiology and pathology of the hearing apparatus. The otologist must think of the hearing apparatus as a complex mechanism, which includes many structures in addition to the eustachian tube, tympanic membrane, middle ear and cochlea. The American Board of Otolaryngology and the schools, therefore, are justified in constantly raising the standards for the younger men.

The author has observed on many occasions a fluctuation in the hearing for bone-conducted sounds. Absence of hearing by bone conduction usually means an organic nerve lesion. Patients with the symptoms of Ménière's disease, however, may have a total loss of hearing by bone conduction at one time and normal hearing by bone conduction at a subsequent examination. So the interpretation of loss of hearing by bone conduction depends on the symptoms of the patient and the other findings on examination.

To make an accurate test of the hearing by either air or bone condition, the opposite ear must be masked. Masking is absolutely necessary if there is a difference in the hearing acuity between the two sides.

It was also shown from a study of patients that tinnitus is not the cause of impairment of hearing by bone conduction. There is considerable evidence which indicates that persistent loud, distressing tinnitus with no other symptoms, a progressive loss of hearing of the inner ear type or a combination of these symptoms with attacks of vertigo may be due to a lesion in the central auditory or vestibular pathways. Certainly, there is abundant evidence to indicate that the lesion which gives rise to Ménière's symptom complex is located somewhere in the nerve pathways and is not due to changes in pressure of the intracochlear fluids or to any local lesion in the temporal bone.

DISCUSSION

E. G. Wever, Ph.D., Princeton, N. J.: The results concerning the neural mechanism of hearing obtained by various experimental methods are more readily coordinated if it is kept in mind that damping in the ear is probably of a high order and that in consequence a single tone is not restricted in its action to a narrow band of the basilar membrane but spreads over a large area. This consideration complicates the theory of the representation of pitch and loudness.

Further experimentation and discussion are desirable on the question of the fundamental character of the cochlear response. The present view is that this response arises from the action of the hair cells of the organ of Corti.

Charles W. Bray, Ph.D., Princeton, N. J.: This symposium is notable for the way in which it has illustrated the extraordinary complexity of the processes involved in hearing. The papers have clearly shown that it is necessary to study hearing by correlated anatomic, histologic, physiologic and psychologic methods.

The necessity for correlated studies greatly increases the difficulties of research, for each of the methods must be applied with due care. Dr. Wilson emphasized in his discussion the precautions necessary in the histologic approach. I do not believe that the possibilities of artefact in the results of the electrical studies have received sufficient recognition. As Dr. Bast pointed out, it is possible to obtain electrical effects similar to the cochlear response from an artificial membrane placed in a solution and exposed to sound. It is easy to show that the membrane is unnecessary. I have obtained similar effects from a simple dish of physiologic solution of sodium chloride by using great intensities of sound and great amplification. These examples illustrate the possibilities of artefact in the use of the electrical method. It is essential that when the electrical method is used a thorough test be made of the association of electrical effects with auditory processes. I believe that when such tests are made the results of electrical studies are of considerable value in analyzing the function of the ear.

There is one other point which I should like to discuss, namely, the statements of Dr. Davis and Dr. Culler on the use of weak stimuli in the study of

the cochlear response. They advocate the use of threshold intensities, and Dr. Davis suggests that the differences between the results of different experiments may arise from the use of different intensities of sound stimuli. This is undoubtedly true, but it seems clear that the important thing is to use a variety of intensities of known physical value. If results vary with intensity, then intensity must be systematically varied before one can understand the results.

Dr. John Tait, Montreal, Canada: It was the wish of Dr. Fowler that some one should present a paper on the function of the saccular macula. He applied to me. This symposium has to do with hearing, and no doubt he believes that in some way the saccular macula is concerned with auditory reception. So do I, but, being unable to present any new facts with regard to it, I had regretfully to decline his invitation.

His prompting, however, did have some effect. It led me to wonder whether any information on the matter might be derived from a subjective study of sound reception under water. As every one knows, cochlear activity can occur in an immobile head. On the other hand, the structure of the saccular macula indicates that it is stimulated by some form of movement—possibly vibrational—of the head itself. As air is a poor energy-conveying medium compared with water, it was at least of interest to inquire whether the quality or range of one's hearing may be modified by plugging the ear holes with wax and then, with an immersed head, listening to sounds under water.

The experiments were conducted in a large city swimming pool. Three normal subjects were used. One could either temporarily immerse one's head or, by means of a specially constructed respiratory apparatus, continue to stay underneath for any required period. Various devices were used for producing the sounds under water at a distance of some 50 feet (15 meters). The result was more surprising than immediately helpful. In every case the hearing was sharper and clearer under water than when the same sound was conveyed by air to the unplugged ears over the surface of the pool! Whether similar experiments conducted on subjects deaf through disease of the middle ear would give more useful information on the matter is for the future to show.

Mr. R. L. Wegel, New York: Being an optimist, I prefer to examine the likenesses that I see in the various papers, and I see plenty of them—enough so that it strikes me that there are a great many things that are known about hearing. It is possible to take the attitude, of course, that one does not know how the ear works, and that is tenable, but if one wants to be completely logical one must also say that it never will be known. I think that the profitable attitude is to emphasize what is known, and if one does that one will be optimistic and continue with new experiments that are useful.

In listening to the papers I am much less disposed to discuss than to ask questions. The first occurred to me in connection with Dr. Davis' paper. I wonder, Dr. Davis, if any plans have been made for measuring the electrical impedance of the various kinds of tissues in the head in order to get an idea of the pathways over which currents generated in the cochlea might be expected to travel.

The next question I have is in regard to Dr. Kobrak's paper. I notice that in making his tests he used rubber tubes, one leading into the outer ear and the other leading into the inner ear, and I was curious to know whether he had made tests on these two acoustic pathways to be assured that their properties of transmission were alike. When tubes are used for conducting sound, there are considerable resonances and transmission differences of as much as 30 or 40 db., depending on the length of the tube and related to the absorption of the sound.

The third question occurred in connection with Dr. Bast's paper. I noticed that the units of measurement given on his slide appeared to be arithmetical, and I wonder if his conclusions would be changed or modified in any respect if he changed to logarithmic units, which are now standard.

Thursday Afternoon Session, May 27

V. ETIOLOGIC AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS"

PRESIDENTIAL ADDRESS: THE DIAGNOSIS OF DISEASES OF THE NEURAL MECHANISM OF HEARING BY THE AID OF SOUNDS WELL ABOVE THRESHOLD. DR. EDMUND PRICE FOWLER, New York.

This address prefaces the clinical portion of the symposium on the neural mechanism of hearing. It is an account of further original observations on the phenomenon observed by me some twelve years ago and reported to the society in 1928. (This was that in certain persons a very deaf ear hears fairly loud sounds quite as well as a normal ear.) The method described is the only one which does not use measurements of air conduction or bone conduction at threshold and does not require confirmation by comparison with bone conduction or by other tests. All the standard tests are frequently uncertain and often misleading, even though their limitations are appreciated. All the tests commonly used for diagnosis and some of their limitations are briefly mentioned.

My test consists of measuring the rate of restoration of the sensation of loudness to its normal state, as judged by the sensation in the opposite normal ear (or in the opposite better ear). This is accomplished by throwing sounds of measured intensity first into one ear and then into the other—in other words, balancing the sensation of loudness in the two ears.

The varying differences in loudness necessary for balance at the various steps above threshold are the measures of the rates of recruitment of the neural elements necessary for equal balance. They are therefore a measure of the location and extent of the loss of nerve elements which initiate, conduct or relay impulses from the end-organs in the cochlea to the cortex of the brain.

Briefly stated, the observations reported last year were:

1. In monaural (or unequal binaural) pure conduction deafness there is no change in the relative sensation of loudness in the two ears at any intensity; i. e., if there is a difference of 30 db. between the sounds which produce the same sensation of loudness at threshold, there will be a difference of 30 db. for all intensities above threshold. In other words, it will take 30 db. (the difference between the sounds necessary to produce the same sensation of loudness in the two ears at threshold) added to the poorer ear to make the sound appear as loud to it as to the better or normal ear.

2. In monaural (or unequal binaural) nerve deafness the poorer hearing ear hears more and more acutely as the intensity of the sound increases, so that, although there may be a difference of even as much as 75 db. between the sounds necessary to produce the same sensation of loudness in the two ears near the thresholds, there may be little or no difference between the sensations of loudness in the two ears at intensities well above the thresholds.

Several apparent exceptions are described and illustrated by graphs. All these are interesting, particularly as demonstrating the pitfalls of the current diagnostic tests and as demonstrating the reliability of the test.

In certain cases there was a lag in the recruitment of loudness, varying directly with the frequency, and this was explained on the basis that the higher the frequency the fewer the nerve elements available for recruitment. This difference in recruitment ratios varies with the type and amount of loss of hearing in the two ears, in other words, with the location and extent of the neural lesion.

In conclusion, a series of graphs were show which demonstrated many variations of monaural and binaural deafness, as shown by air conduction and bone conduction at minimum audibility and as shown by my test with sounds well above minimum audibility.

Certain interesting corollaries are set forth, which give a better understanding of the phenomenon discussed. It is shown how to differentiate tinnitus originating from the middle ear from that arising from the inner ear and how the test is useful to detect malingering. The test does not require any masking of either ear.

TRANSIENT OR VARIABLE NERVE DEAFNESS FROM CIRCULATORY EFFECTS: HIGH AND LOW INTRACRANIAL BLOOD PRESSURES. DR. ALBERT C. FURSTENBERG, Ann Arbor, Mich.

NERVE DEAFNESS WITH VERTIGO AND TINNITUS (MÉNIÈRE'S SYMPTOM COMPLEX). DR. CLARENCE H. SMITH, New York.

The term Ménière's disease is a misnomer. No distinct entity gives rise to paroxysmal vertigo, tinnitus and deafness.

Cases of toxic auditory neuritis are not to be classed as instances of Ménière's syndrome. Ménière's complex does not always follow the same pattern. In some cases it is caused by obstruction of the eustachian tube, in some by increased intralabyrinthine pressure from within the cranium and in some by an excess of water or of sodium chloride in the labyrinth, while in others it is caused by physical changes in or around the vestibular branch of the auditory nerve.

The constituents are deafness, generally unilateral, of the type involving the perceptive mechanism; tinnitus, unilateral and much exaggerated during paroxysms, and vertigo, sudden, sometimes nocturnal and generally lasting three or four hours.

Treatment in Different Types.—Unilateral obstruction of the eustachian tube can be cured by local measures, persistently carried out. The common means are inflation, the use of the bougie and intranasal treatment.

Furstenberg's treatment by diet, in which he limits the sodium intake and administers ammonium chloride internally, helps in a certain proportion of cases.

Patients whose condition is dependent on abnormal arterial tension are aided by the use of cardiac stimulants or depressors.

Dandy reports torsion of the auditory nerve by the encircling pressure of large arterial branches. In these cases Ménière attacks were present.

Dandy, Coleman, Cairns and McKenzie have operated on patients with Ménière's symptom complex and have reported almost uniform success. The operation consists in the severance of the vestibular branch of the auditory nerve. Portmann's operation consists of section of the saccus endolymphaticus to relieve the intracranial labyrinthine pressure.

DISCUSSION

DR. LESTER M. HUBBY, New York: An understanding of why it is that in association with hemorrhage or effusion into one or both labyrinths, tumor formation near the vestibular nerve, etc., in one series of cases there will be slight if any attacks of vertigo while in another series there will be frequent and violent attacks requires a study of the basic modus operandi of vertigo.

Continuous nerve impulses travel to the brain centers from the vestibular receptors in the two labyrinths. The equilibrium centers at one period may be receiving either equal and normal centripetal nerve impulses from the labyrinths or may even be receiving no impulses from one labyrinth and few from the other and yet there may be no vertigo. In other words, a state of compensation is always being reached for the number of impulses received.

Any sudden sufficient change in these impulses from one labyrinth, whether an increase or a decrease of nerve impulses, results in an upset of the equilibrium in the vestibular centers, nystagmus and then a sense of vertigo in the higher centers, perhaps accompanied by somatic reactions, such as nausea, vomiting, profuse perspiration, pallor and fainting, depending on the violence of the reaction.

That this disturbance of equilibrium is easily produced in the vestibular neurons near the ears is shown by the response to caloric or galvanic stimulation, to turning or to the fistula test or by suddenly destroying one labyrinth or cutting one vestibular nerve near the internal auditory meatus.

The character of the vertigo is determined by the vestibular neurons affected.

If the system of neurons leading to the crista acustica is affected, the vertigo will be rotary; if the systems leading to the maculae acusticae of the utricle and saccule are also involved, the vertigo will be the result of the combined effects.

The utricular disorder is supposed to cause sudden falling down with relaxed muscles, while the falling caused by a saccular disturbance is to one side.

If there is great dissimilarity in the function of the two vestibular systems, so that the vestibular centers are largely dependent on the impulses from one side, only a moderately sudden change of these impulses can initiate vertigo.

The vestibular system of neurons is the most important part of the apparatus of equilibrium and should not be destroyed except when it is absolutely necessary.

WORD DEAFNESS (CONGENITAL). DR. PAUL DOZIER, Philadelphia.

Congenital word deafness is a selective difficulty in the recognition of spoken words which exists from birth. There are resultant delay and distortion in speech. The hearing is perfect, and the interpretation of meaningful sounds other than speech sounds is unaffected. The language difficulty is more apparent as the syllable and word sequences become longer and more complicated. The defective speech is usually of the infantile type, with substitutions, elisions, unevolved consonants and their combinations, etc. The scanty vocabulary contains infantilisms and neologisms. Except for motor overactivity and a tendency to delayed or mixed evolution of handedness, there is no neurologic defect. Standard psychometric tests show a comparative inferiority in situations dependent on understanding or use of the spoken word but otherwise suggest potentially normal intelligence. Problems of emotional adjustment are present and may increase the delay in function, but no evidence points to emotional causation.

Word deafness should be distinguished from regional deafness and from general intellectual defect. The audiometer differentiates the former, although informal tests are helpful with children too immature for audiometry. The latter is excluded only after most complete psychometric studies and learning trials. The difficulty is distributed in all degrees among different persons: In the preschool child, the speech disability and questionable hearing are presenting features, while the child of school age, whose handicap is less severe, may present auditory inattention, poor speech, reading difficulty and the failure in school work of an intellectually retarded child. Orton, in this country, and Ewing, in England, have recorded their cases most accurately. Experiments in retraining are as yet limited, but experience with the comparable condition of congenital word blindness suggests that special teaching methods are eminently worth the trial. No evidence of a progressive pathologic process is present, and the condition is regarded as a constitutional variant, comparable to congenital word blindness. It may be connected, like the latter condition, with failure to establish complete and efficient language function in a single cerebral hemisphere.

WORD DEAFNESS (ACQUIRED). DR. EARL C. CHESHER, New York.

The purpose of this paper is to describe the symptoms of word deafness as they are seen in patients with neoplasm of the temporal lobe in the dominant cerebral hemisphere and to relate these findings to the larger subject of acquired language defects, commonly called "aphasia." In the course of examining over 300 aphasic patients admitted to the New York Neurological Institute, I did not observe a single case of pure word deafness. A method is described briefly which permits a quantitative estimation of the percentage of reduction in each part of the language mechanism, including the understanding of the spoken word. It is shown that word deafness occurs as a part of sensory aphasia and that it is rarely, if ever, the greatest language defect present even when the pathologic involvement is limited to the temporal lobe. Seventeen patients with verified primary tumor of the temporal lobe are described. The types of error made in response to the spoken word are tabulated, and examples of typical responses are given. The defect in understanding of the spoken word is compared with other defects in language—naming, repetition, reading aloud, understanding the printed word, copying, writing from dictation and spelling. The spontaneous speech of these patients is described, and examples of typical responses are given. Language profiles are given to illustrate that while word deafness does not occur in the absence of other language defects there are frequent examples of an

aphasic state in which there is no word deafness. Cases are cited of pure motor aphasia without defect in the understanding of the spoken word. It is shown that in these cases the lesion did not encroach on the temporal lobe, and it is suggested that when word deafness is a part of the aphasic state this area is involved by the lesion.

PSYCHOGENIC FACTORS (HYSTERICAL AND EMOTIONAL). DR. MILES ATKINSON, New York.

Otologists do not take sufficient cognizance of the psychogenic aspect of hearing. One of the most important factors is that of adaptation. If there is any interference with hearing, the degree of compensation depends on the patient. A child will compensate readily for loss of function. An old man, beginning to grow deaf possibly through senile neuritis, has difficulty in hearing conversation although the diminution of hearing as measured by audiometric tests is apparently not great. He hears simple sounds, but he is unable to make these sounds into the pattern of speech.

Psychogenically, the auditory tract may be divided into the external organ of hearing and the centers in the brain which perceive simple sounds and the higher cortical center of intellectual hearing which converts these sounds into the pattern of speech.

The first, "the basic hearing," is what is tested. The second is intellectual, and the ability to hear intelligently, while it depends on the degree of deafness, varies in different patients. The old man cannot adapt himself. He cannot become accustomed to hearing the word "ship" as "fip," and getting the meaning. There are persons who have a better flair for learning language than others.

There is another psychogenic factor in conditions called "functional hysteria." Many patients with this condition were seen during the World war. They are persons with undisciplined minds. They have unstable nervous systems which are unable to withstand insult:

> "What with noise, and fear of death,
> Waking, and wounds, and cold,
> They filled the cup for my Mother's son;
> Fuller than it could hold."

The hysterical person is not acting purposely. He really believes that he is deaf. The malingerer can be caught by a trick, and the hysterical person can be cured by sufficient force of personality and by reproducing the localized subject causing his functional disorder.

The malingerer knows that he is not deaf. He has a motive in simulating deafness—to avoid going to sea or to the trenches or to get remuneration.

One such malingerer was put in the hospital for observation. I went around with the orderly every day and ordered all kinds of delicacies on the chart—cream with stewed fruit one day, chicken another day—but instructed the orderly to give the patient nothing but milk. We talked in an ordinary tone, and the man pretended to be very deaf. Every day the orderly would say to me: "Yes, he had that and enjoyed it very much." The patient stood this for about a week. One day the orderly said: "Oh, he had a fine dinner yesterday; he enjoyed it." I replied: "That is very good." The patient said: "It's a bloody lie. I have had nothing but milk for a week."

DISCUSSION

DR. L. VOSBURGH LYONS, New York: "Psychic deafness" is an unconscious rejection of sound by the psyche.

I shall recall a few types of psychic rejection of sound that all must grant are normal. In a crude way let me compare hearing with vision. In vision there is a center of macular clarity and around that an aura of much more obscure vision which, although it produces an effect on the reactions, seldom reaches clear consciousness. So with hearing, there is a central point of clarity on which one focuses the attention, and there is an aura of sounds which one ignores or rejects. The ears of the audience are at this moment assailed by a great mélange

of sounds, but I trust that there is only one particular group of them on which they are bestowing their attention.

In vision, the clarity is shifted by an actual movement of the eye, and special anatomic structures mediate the clear impressions. No such structures that determine clarity of hearing are known, and otologists say that the shift is determined by "attention." One can listen to the same phonograph record twice. The first time one focuses the attention on the trumpets. The second time, from exactly the same stimulus, one can focus the attention on the percussion instruments and be oblivious of the trumpets. This consciously determined rejection of sound is a normal phenomenon.

I shall go one step further. The example that I am about to give is on a much higher level of integration. I know a man who does not hear his wife when in ordinary conversation she asks him to do chores around the house. It is obvious that part of his psyche must comprehend the nature of the request, because he always hears her when she suggests going to the movies. One can say in this case that the rejection is on an emotional basis.

If one knows the man and likes him, one will say that the mechanism is within the normal limits and not to be classified as pathologic. It certainly embodies the elements of a psychoneurotic mechanism.

I shall touch on obviously hysterical deafness. Unilateral rejection does not present a serious diagnostic difficulty. Many hysterical persons regard half of the body as bad or diseased. This is observed frequently after alleged trauma in cases involving litigation. There may be hemianesthesia or hemiparalysis, and many times the patient claims to be unable to hear on the "bad" side.

There is hysterical selective deafness in which the patient cannot hear under a given set of circumstances. He may be unable to hear in a certain house or he may be unable to hear when a certain person speaks to him. Obviously, there is no diagnostic difficulty with deafness of this type.

There are cases in which there is total rejection of all sound, and these may present considerable diagnostic trouble. Dr. Fowler Sr. has mentioned loudness balance tests which have made the differentiation in some cases. Without such a test, the diagnosis could be made only after a rather thorough study of the total personality. Many times one can discover the purpose that this deafness serves. In the World War hysterical deafness kept the victim away from the firing line. Many times in civil life the purpose is not as obvious as that. There is another factor which is of great value in the differential diagnosis, and that is an evaluation of just how much the patient is willing to do to compensate for his affliction. A truly deaf person will study lip reading and make all sorts of compensatory efforts. As the hysterical deafness serves some sort of purpose, the patient will not make too much effort to lose it, although he may go through the motions of consulting physicians. After getting to know the patient well, the physician can detect these weak spots, although the more intellectual the patient, the more water tight the case that he can concoct.

The nature of the therapy must vary with the nature of the condition and, I might add, with the temperament of the therapeutist. Dr. Atkinson has mentioned patients who got well when a sudden stimulus was applied. Unfortunately, this type is much in the minority and the circumstances are usually fortuitous. Some intellectual persons can be made to understand the nature of the psychic mechanism causing their symptoms and to overcome them by the rational method. However, the majority of human beings, especially neurotic persons, cannot be approached by rational methods.

Physicians know that many persons are benefited by the mystic approach. The happenings at Lourdes bear witness to that. Most sincere physicians will not stoop to what they call "bunk," with the result that the mystic approach has gravitated into the hands of charlatans. Many of the "cures" are conducted on a mass basis, which allows the patients to work one another up to the pitch necessary for the mental change to take place. This can hardly be done in the privacy of a physician's office, and I feel that members of the medical profession should give more thought to this matter.

There are personal factors, however, that the physician can and does make use of. As soon as the patient begins to like and trust the physician, he can be appealed to not to let his doctor down. If he does not get well, it will be discreditable to the doctor; therefore, he must do everything he can to bring credit to the man who has done so much for him. Even the psychoanalysts are willing to admit that this mechanism plays a great part in the rehabilitation of many neurotic persons.

MALOCCLUSION AND ITS RELATION TO AURAL AND TEMPOROMANDIBULAR DISORDERS. WILLIAM H. CRAWFORD, D.D.S., New York.

The loss of permanent teeth frequently results in malocclusion and shortening of the space between the upper and the lower jaw. This in turn sometimes brings pressure on nerves and other structures in or near the joint of the lower and upper jaws. As a result of these conditions, patients complain of a large variety of trouble. Pain in and around the joint and ear are most common. Clicking of the jaw on opening, and buzzing and rasping noises in the ear are also common. More serious symptoms are dizziness and loss of hearing. All the aforementioned conditions may be traced at times to overclosure of the jaw and therefore may be corrected by placing in the patient's mouth suitable appliances to correct the opening between the jaws and the relation of structures in the mandibular joint.

DISCUSSION

DR. GEORGE M. COATES, Philadelphia: My interest in this problem dates to the work done by Dr. George Fetterolf in conjunction with Dr. David Goodfriend. That study was undertaken to try to determine whether there is any etiologic relationship between malocclusion and impairment of hearing, tinnitus and vertigo. While the study was incomplete, it seemed that at least enough results were obtained to justify the feeling that in certain cases such a relationship might exist. The recent careful studies of Costen are related to this problem, although they apply principally to the production of pain as a result of malocclusion causing closure of the bite, with corresponding changes in the normal relationships in the temporomandibular articulation. From the large number of patients with some degree of malocclusion mentioned by Dr. Crawford, it would seem obvious that this factor as a cause of impairment of hearing and associated symptoms cannot be common. Otologists know definitely that there are other common causes, such as focal infection, acute infectious disease in childhood, infection of the nose or throat, traumatism, drugs, occupation, senility, etc., which will account for the symptoms in a majority of the cases. Nevertheless, it is important to keep in mind the possibility that dental disorders, other than infection, may cause impairment of hearing, tinnitus and vertigo, especially in cases in which other factors are not in evidence, although these symptoms may occur only in a small proportion of the cases. It is for this reason that the subject is of interest and that further clinical research should be encouraged. It is especially necessary that there shall be close cooperation between the departments of dentistry and otology if results are to be reliable.

Whether the bite shall be opened will depend on individual circumstances. It may not be useful in cases in which the dental cups have become flattened because of excessive use, but when the key teeth have been lost, restoration of the normal bite may cause alleviation of some symptoms, notably pain. I believe that I have seen this happen in some instances. I agree with Dr. Crawford that other, more usual, means should be tried first, but that need not throw the consideration of the bite too far into the background. If opening of the bite is not detrimental to these persons, the only objections to a trial of this method are the discomfort to the patient, which is not great if the work is skilfully done, and the expense. If the mouth is edentulous, the question is not so complex and the chance of doing damage to the supporting structures is nil. Much more light is needed on this subject, and if opening the bite is to be attempted, it should be done after careful measurements, as suggested by Goodfriend, and in conformance with well worked-out anatomic and mechanical principles.

Friday Morning Session, May 28

SIMULATED DEAFNESS (MALINGERING). DR. DOUGLAS MACFARLAN, Philadelphia.

An intimate knowledge of the tests for malingering is essential to the otologist, not only for the occasional testing of the true malingerer (for one sees few of them) but for the sake of accuracy in the reporting of the true condition of the hearing of other patients, who may be deceiving themselves and the physician by guessings, imaginings and tone memories. The better known and commonly used methods and tests are described. A test for the detection of the degree of residual hearing in the deaf-child-before-language is described, and the subject of determining attention deafness is discussed.

DISCUSSION

DR. JOSEPH C. BECK, Chicago: The experience that all otologists have had in determining whether a person is simulating unilateral deafness or really is deaf shows that it is comparatively easy. It is the person who is completely deaf or who says that he does not hear at all with whom one has the great difficulty, and all tests fail in many respects in these cases.

Dr. Macfarlan mentioned the detection of remnants of hearing in the young child, and there is an old test which Bezold invented. He found that the obnoxiousness of a sensation in comparison to the pleasantness of a sound will react on the child when one puts the fork on the vertex or on any other part of the head. If the child is deaf, he will resent the sensation, whereas if the sound is musical, as is that made by the fork, he will smile, unless he is frightened by the fork, as many children are.

I am connected with an institution where my associates and I have to examine malingerers. Most otologists have had similar experiences in civil practice, in institutions, in labor organizations and so forth when deafness was simulated because it meant an economic advantage to the person to have it determined that he was deaf. The professional educator for the simulator is at hand; men have learned how to educate others to show that they are deaf and how to react to tests so that it becomes a difficult matter to determine whether they are really deaf or are simulating. Professor Zaufal taught me a test for which I am sure most otologists have had the instrument although it is difficult to get in certain parts of the country. The simulator walks along on any pavement that will resound, not knowing that any sound is about to be produced. The examiner drops a silver dollar; it makes a sound, and the malingerer will turn around. It is one of the good tests that Professor Zaufal taught me.

The test of Bárány and Lombard—raising the voice when reading, while making a noise in the opposite ear or in the supposedly hearing ear—is good but only in cases of unilateral deafness.

It is surprising how wonderfully the simulators become educated to the finest tests that are known. That physicians cannot control.

Dr. Macfarlan called attention to a test of the vestibular apparatus associated with a test of the auditory apparatus. All otologists know that those are two entirely separate functions; yet in most of the pathologic conditions within the channels or the fluid of the internal ear, any process that affects one is likely to affect both, and the result of a vestibular test will indicate the extent of real involvement. There is an ocular test associated with an aural test. A sharp-sounding fork is placed before the supposedly deaf ear with the other ear completely masked. If it is struck sharply beside the ear, the pupil will contract. That cannot happen if the ear is really totally deaf, but what about partial deafness? One has to decide the degree of deafness.

While the evidence of the lie detector is not admissible in court, it is an excellent device to indicate for one's own knowledge when a patient is simulating or actually lying.

There are truly hysterical patients who have a certain degree of attempted malingering for some reason or other, and there is the rub. Who can recognize

the patient who is not to blame or does not know to what his hysteria is due? One usually finds when a patient is hysterical that he has hysterical manifestations in other parts of the body, and one can usually find those, but they can also be simulated. The malingerer simulates not only deafness but other things.

It is important to go into the story of the simulator, but when economic stress is present, $60.50 a month additional will play an important part in making him a wonderful simulator, and it will take the best of physicians to catch him even after thorough testing.

The lie detector is an advance, and through the kindness of the gentlemen in charge of this apparatus at Northwestern University, I was privileged to make use of the instrument in my tests. It will be a great aid in the detection of malingerers.

NERVE DEAFNESS FROM FAMILIAL AND DEVELOPMENTAL DEFECTS. DR. MAX A. GOLDSTEIN, St. Louis.

A classification of congenital deafness was suggested by me in 1921: (*a*) biologic congenital deafness, which depends on biologic and anatomic irregularities in growth as they may affect the temporal bone; (*b*) pathologic congenital deafness, which depends on the pathologic changes that may take place during fetal development or at the time of birth and includes hereditary syphilis, rickets, quinine toxemia, intra-uterine hemorrhage and birth injury.

The concluding paragraph recites the relationship of early otosclerosis and its differentiation from other types of pathologic congenital deafness. In the laboratory the presence of otosclerosis has been determined in a child 18 months of age and therefore the possibility of its development in the fetus must be considered.

DISCUSSION

CHARLES B. DAVENPORT, PH.D., Cold Spring Harbor, N. Y.: Two things have become clearer in the study of genetics in the last twenty-five years, and these may well be applied to man. One is that the simple mendelian formula of single factors that are either recessive or dominant has been largely replaced by the discovery that many apparently single traits are really due to the cooperation of two or more factors, so that a simple mendelian interpretation of their occurrence cannot be found.

Years ago I started to analyze the data concerning the inheritance of congenital deafness, as set forth in Fay's "Marriages of the Deaf." It soon appeared that when both parents were deaf-mutes frequently all the children could hear normally. At that time it was determined that this must be due to the fact that the number of developmental elements in the auditory apparatus was so great (the loss of any one of them might cause complete deafness) that in different cases deaf-mutism was due to the absence of different factors. The defect from the germ cells of one parent might be covered up in the offspring by the absence of that same factor in the other deaf-mute parent, who was deaf-mute because of a different factor. This hypothesis is probably entirely justified in many cases. It seems to be supported by the fact that when the parents were deaf-mutes and cousins, all the children were usually deaf-mutes. This is because members of the same family probably are deaf because of defects in the same units of structure. However, in the lapse of years a somewhat different interpretation has become available.

Secondly, a striking result of genetic study has been the discovery that what is apparently a single homogeneous trait in the population often is genetically diverse. Thus, Dr. E. C. MacDowell, of Cold Spring Harbor, N. Y., has for many years bred a strain of waltzing mice. In the course of years, different types of waltzers appeared. Though the resemblance of these was manifest to the casual observer, the different types proved on cross-breeding to be genetically distinct. When two waltzers of distinct strains were crossed, instead of giving, as such matings inside one strain give, only waltzing offspring, they gave only normal offspring.

Similarly, MacDowell has been rearing many strains of mice with leukemia. These have all descended from one female. He has many lines of descent of these leukemic mice, which are so similar in their symptoms that it would be impossible to separate them into different categories were their pedigrees not known. Two leukemic mice of the same strain tend when mated to produce only offspring which will die of leukemia if they live long enough. However, when leukemic mice from two distinct strains are crossed, the offspring are not ordinarily susceptible to leukemia. Thus, a pathologic trait which is single and uniform by every phenotypical criterion proves to be diverse genetically and to behave in inheritance in a diverse fashion. It has been shown for a number of pathologic traits in human beings that they are genetically diverse, and since this genetic difference implies a chemical difference, it may well be that such diverse strains would require dissimilar clinical treatment. Indeed, it seems probable that within the next fifty years the textbooks on pathology will have to be rewritten, that each of the old pathologic entities will be broken up into several or many, perhaps in the same way as pneumonia, in which there are now recognized many different types which require special treatment.

It would seem that in the future the pathologist must work hand in hand with the geneticist to get an understanding of his problems.

Occupational Deafness (Traumatic): An Audiometric Study. C. C. Bunch, Ph.D., St. Louis.

My study is an attempt to determine more specifically the nature of the deafness which occurs as a result of occupational influences. I first examined the textbooks and the reports of earlier investigators to determine whether there are characteristics which make it possible to distinguish occupational deafness from that which has some other cause.

Audiograms with detailed histories of a large number of patients who have been working as boiler makers, machinists, gunners and sportsmen, telephone operators, caisson workers, etc., were shown. Admitting that in no case has a definite cause and effect relationship between acoustic insult and deafness been proved, I am led by the similarity of the records together with the evidence from the literature to the following conclusions:

1. If a loss of auditory acuity has resulted from the effects of excessive stimulation by industrial noises, it is first evidenced by an abrupt dip or gap in the range of hearing as determined by the audiometer, usually near ¢-5. Acoustic trauma should be suspected when these gaps appear.

2. With continued excessive stimulation, this gap in the curve becomes deeper and broader to include the tones above and below ¢-5. Eventually, the extremely high tones of the audiometer become inaudible and the curve shows an abrupt drop, usually between ¢-4 and ¢-5. Many times there is a tendency for the curves to approach the normal line at ¢-6, with the deepest point in the gap near ¢-5. If the loss does not extend downward to include ¢-4, these persons may note little loss for the spoken voice.

3. The perception of low tones is least often involved. The loss for low tones is never as great as that for high tones.

4. The greatest loss consistently appears near ¢-5. Often there will be little or no loss for ¢-4. If the tests are made with tuning forks rather than with audiometers and the ¢-5 fork is not included in the set, many of these losses will not be discovered.

5. If the trauma to the ear results in the rupture of the tympanic membrane with subsequent otitis media, the contour of the hearing curves will be entirely different from those in cases in which the deafness results from occupational noises alone. The same is true of the deafness of caisson workers, which presumably is caused by the formation of bubbles within the cochlea.

I have prepared a bibliography of 160 titles.

DISCUSSION

DR. JAMES G. DWYER, New York: Dr. Bunch is to be congratulated on his thorough and exhaustive report on occupational deafness. I feel capable of discussing caisson disease only as it affects the ears. Years ago I had an opportunity of examining at autopsy persons with the "bends," as this condition was then known. When the Thirty-Fourth Street tunnel was being constructed, I observed many cases. At autopsy I would find bubbles of air in the spinal fluid, heart and blood vessels and many hemorrhages in the brain. The air would be driven into the blood vessels, and when sudden decompression occurred the same result ensued in the body as when one releases a syphon of soda. Hence, the rationale was that hemorrhages in the labyrinth and the organ of Corti resulted in deafness. Five years ago a patient came under my observation with the history of having had an attack of caisson disease two months previously. He was totally deaf and very dizzy; yet caloric and rotation tests produced no nystagmus. In spite of the absence of nystagmus, the patient became terrifically dizzy. This case was reported at a meeting of the American Otological Society some years ago.

ATROPHY OF THE ACOUSTIC NERVE AND END-ORGAN FROM ROENTGEN RAYS AND RADIUM. DR. V. Y. KASABACH, New York.

This paper is a preliminary report on the effect of roentgen rays on the hearing. It includes the report of 9 cases of neoplasm in the nasopharynx or near the region of the ear. In treatment of the primary lesion with intensive radiation of from 2,000 to 4,000 roentgens, the ears had to be included in the field of irradiation. Comparison of the audiometric readings taken before the course of roentgen therapy with those repeated at weekly, bimonthly and monthly intervals showed improvement in the air and bone conduction in 6 cases. Three patients with advanced neoplasm associated with anemia and inanition showed a decrease in the acuity of hearing with failing health, even though there was initial regression of the growth after roentgen therapy.

It is reasonable to assume that the primary site of the tumor, causing obstruction to the eustachian orifices, may have a bearing on the impairment of the hearing. After a course of radiotherapy the tumor decreased in size or disappeared, relieving the compression against the eustachian tubes and producing a notable improvement in the readings for air conduction. The readings for bone conduction also improved simultaneously after roentgen therapy. It is too early to evaluate the significance of this observation. However, it is reasonable to assume that intensive radiation may have a direct effect on the nervous mechanism of the auditory system. A similar investigation of patients with partial or complete nerve deafness might throw further light on this interesting subject.

In other instances the acuity of hearing varied with the general health and well-being of the patient during the course of radiotherapy. The hearing was impaired when the patient complained of "radiation sickness" or of general weakness, but improvement took place soon after the cessation of these complaints.

These observations lead me to believe that high voltage radiation applied to the auditory system may be of therapeutic service in selected cases.

DISCUSSION

DR. DOUGLAS QUICK, New York: A review of the literature confirmed my impression that nothing published before 1920 on the effect of roentgen rays on hearing would be of value, since roentgen therapy prior to that date was largely empirical. In a review of the more recent literature, it is interesting to note that when benefits are claimed, the dose, whether of radium or of roentgen rays, has been very small. This refers particularly to the benefits reported from the treatment of tinnitus, to which Dr. Kasabach referred in the opening of his paper.

The point of view of those dealing with malignant disease differs of necessity from that of the otologist, since in the treatment of malignant disease possible

damage to special sense organs must be a secondary consideration. This being the case, patients who would come under their immediate observation would be those who have received heavy and prolonged irradiation.

The group of cases reported by Dr. Kasabach falls into this classification, and he has noted that in patients showing therapeutic benefit from the treatment of cancer, there was an improvement in the hearing likewise. They have been followed, however, for only about six months. It will be exceedingly interesting to note his observations on later follow-up, and it is my surmise that atrophic changes will appear much later, if one may judge from the experience with many patients with a growth in the nasopharynx, who have been well of their malignant disease for long periods. While the state of hearing of these patients has no accurate check, I have been impressed, since being asked to participate in this discussion, on making follow-up inquiry concerning persons who showed no evidence of cancer to find a substantial number showing progressive deafness. They have all had quantitatively heavy, as well as heavily filtered, irradiation.

If one may speculate a bit further and draw on the experience of those who have been interested in the employment of irradiation for the treatment of acute or subacute inflammatory processes in general, one may conclude that in the long run improvement in such conditions as tinnitus, Ménière's disease and some of the persistent inflammatory conditions will probably be obtained with small doses, not sufficient to produce atrophy.

NERVE DEAFNESS FROM TOXIC SUBSTANCES. DR. ELLISON L. ROSS, Chicago.

Diphtheria toxin of 100, 50 and 25 per cent strength, with a minimum lethal dose of 0.0042 cc., was injected into the middle ears of dogs and kept there for one hour. The strongest concentration destroyed the vestibular functions on the side into which it was injected, and microscopic examination revealed much damage to the cochlea. Weaker concentrations of the diphtheria toxin destroyed the vestibular function in most cases.

Arsphenamine and neoarsphenamine in a concentration of 1 per cent used in the same manner as the diphtheria toxin destroyed vestibular function in every instance. Microscopic examination of the ears from nine to twenty-three days after the injection of arsphenamine showed marked injury to the nerve cells and tissue cells of both the ampullae and the organ of Corti.

Concentrations of the arsphenamines of 0.5 per cent destroyed the vestibular function in the majority of animals, but a 0.1 per cent solution had no permanent effect.

Salyrgan in 5 per cent strength applied to the middle ear destroyed the vestibular function. When it was used in a 1 per cent concentration, no effect was observed.

Quinine hydrochloride in 10 per cent strength only temporarily depressed the vestibular function. The same was true of a 5 per cent solution of phenol; apparently both act as anesthetics.

Methyl alcohol in 10 per cent strength in the middle ear had no effect on equilibrium.

DISCUSSION

E. A. CULLER, PH.D., Urbana, Ill.: Using Dr. Ross' technic, my associates and I injected diphtheria toxoid into the middle ear to determine its effect on the thresholds of hearing in the dog. Having first destroyed one ear, we obtained normal thresholds for the remaining ear at 125, 1,000 and 8,000 cycles; then we injected 1 cc. (1 part of toxoid to 3 parts of a physiologic solution of sodium chloride) into the middle ear and measured the loss in acuity. At 1,000 cycles the animal lost a smaller fraction of its total range of acuity than at 8,000 and a greater fraction than at 125; in other words, the proportional effect of the toxin is greatest at 8,000, next at 1,000 and least at 125 cycles, as is to be expected from the topographic distribution of the hearing mechanism along the basilar membrane.

NERVE DEAFNESS FROM DRUGS AND CHEMICAL POISONS. DR. H. MARSHALL TAYLOR, Jacksonville, Fla.

Drugs may have a definite toxic action on the ganglion cells of the cochlea and the associated nerve fibers. Drugs may cause changes in the endothelium of the smallest capillaries or may cause a contraction of the blood vessels of the internal ear, with ischemia, lack of nutrition and anoxemia, resulting in degeneration atrophy of the ganglion cells and the nerve fibers in the basal coil of the cochlea. They may also cause a diminution in the endolymphatic pressure, with resulting collapse of the membrana Corti.

Drugs may injure the cortical center of hearing, some having an affinity for the cytoplasm of the cells while others may have an affinity for the nuclei. Recently, I had spinal punctures made on patients who had been given 30 grains (1.94 Gm.) of quinine for malaria and was interested in finding quinine in the cerebrospinal fluid. The presence in the cerebrospinal fluid of such agents as quinine, the salicylates and alcohol is not without clinical interest, and its importance has, perhaps, yet to be evaluated.

Quinine.—Of all the drugs which cause deafness, quinine is the most important. It has a definite toxic action on the cells of the spiral ganglion and seems to have a special affinity for the cells in the basal turn of the cochlea. Quinine acts not only as a protoplasmic poison but as a vasoconstrictor. The resulting ischemia, lack of nutrition and anoxemia may cause degeneration atrophy of the ganglion spiralis and Corti's organ. Quinine may induce hypotonia of the labyrinth and a diminution in the endolymphatic pressure, with resulting collapse of the membrane of Corti.

Quinine is frequently used therapeutically in disease concurrent with pregnancy, as an abortifacient and in the induction of labor. There is ample evidence that the blood of the human fetus under such circumstances may be cinchonized, for quinine is found in the urine of the new-born baby and repeated autopsies on infants whose deaths were attributed to quinine given the mother in the induction of labor have shown quinine in concentration in the brain and liver. The human placenta acts as a dialyzer of drugs, and crystalloid substances, such as quinine, readily permeate the placenta and are found in relatively the same strength in the fetal circulation as in the maternal circulation. Experimentation with animals has shown that quinine in the fetal circulation produces the same pathologic changes in the ganglion spiralis and the organ of Corti that quinine does in the adult ear.

Quinine used in the early months of pregnancy in diseases concurrent with pregnancy or as an abortifacient may damage the ganglion cells of the fetal ear.

Quinine should be prescribed with the utmost caution to persons who have a hereditary tendency to deafness and to early atrophy of the auditory nerve. Quinine is definitely contraindicated in the case of the pregnant woman who has a family history of deafness.

Salicylates.—Many persons are highly sensitive to the action of the salicylates and on taking them may have hissing and roaring sounds in the ear or deafness. The chief pathologic change in the auditory apparatus in poisoning from the salicylates is found in the nerve cells of the spiral ganglion. The salicylates readily pass into the fetal circulation when administered to the expectant mother. The possibility of their prolonged administration affecting the fetal ear should not be ignored.

Alcohol.—Deafness has frequently been attributed to alcohol. Alcohol is one of the drugs which readily pass into the cerebrospinal fluid. In most cases of deafness from alcohol the onset is sudden, deafness frequently developing within a few hours.

Alcohol is also transmitted through the placenta when it is administered during pregnancy. This phenomenon cannot be ignored when one considers the etiology of deaf-mutism in the large number of cases in which it has been attributed to chronic alcoholism in the mother.

Arsenic.—Deafness and tinnitus have often been reported after the continued use of arsenic. Experimentation with animals following the administration of arsenic has shown definite changes in both the vestibular and the cochlear branch of the eighth cranial nerve. Arsenic is one of the drugs which affects not only the ganglion cells but Reissner's membrane, which may change the endolymphatic pressure.

Tobacco.—Tobacco sometimes causes nerve deafness, with associated disturbances of equilibrium. Tobacco has been shown by experimentation with animals to have a selective action on the cells of the spiral ganglion.

Other Toxic Substances.—The effects on the hearing of other substances, such as carbon disulfide, oil of chenopodium, mercury, morphine, aniline dyes and insulin, are described in detail.

DISCUSSION

DR. BENJAMIN P. WATSON, New York: For more than twenty-five years my associates and I have used quinine in the hospital and in private practice for the induction of labor. Until ten years ago our routine was to give the mother 10 grains (0.65 Gm.) of quinine hydrochloride in solution at two hour intervals, for three doses—30 grains (1.94 Gm.) in all. In the past ten years we have given three 5 grain (0.32 Gm.) doses in a capsule at the same intervals—15 grains (0.97 Gm.) in all. After the 30 grain (1.94 Gm.) total dose in solution, many of the mothers complained of ringing in the ears. With the 15 grain (0.97 Gm.) total dose in capsules this is the rare exception. In no case that has come under my observation has there been any complaint of a permanent ill effect on the auditory apparatus of the mother.

There are persons who have an idiosyncrasy to certain drugs, and they may react in different ways. During the World War I saw service in Macedonia, where malaria was prevalent and of a malignant type. Every one was ordered to take quinine as a prophylactic, but certain nurses in the unit could not do so because of the severe uterine cramps it caused. They did not suffer from tinnitus.

The work of Dilling, Gemmell and Sadler (*J. Obst. & Gynaec. Brit. Emp.* **36**:352, 1929; **37**:529, 1930) showed conclusively that quinine does pass from mother to child in utero, that it is excreted in the fetal urine and that it may cumulate in the fetal tissues. Their conclusions as regards the risks to the life of the child from the induction of labor by quinine are as follows:

". . . We have collected the records of 805 cases of quinine induction and, accepting six as the number of deaths probably attributable to quinine, the percentage of death-rate due to quinine is 0.75 per cent. If doubtful cases are included we find 11 deaths, or 1.36 per cent. For comparison we have analysed the number of foetal deaths from undiscovered causes occurring during a series of 2,682 normal labours: these amount to 36 in number which represents an undiscovered death-rate of 1.34 per cent. There can be no doubt, therefore, that although scientific evidence is available that there is a risk involved to the child when induction by quinine is employed, this risk, from the practical point of view, is so small that a labour induced by quinine is at least as safe as an unassisted delivery."

With this conclusion I, from my own experience in a large number of cases, am in entire agreement.

Dr. Edmund P. Fowler Jr. undertook to analyze the charts of all the mothers who had received quinine for the induction of labor in the Sloane Hospital for Women and to follow, as far as possible, the babies born of these mothers. This follow-up was possible, as there is the closest cooperation between the Sloane Hospital and all the clinical departments in the Medical Center.

Dr. Fowler Jr. has gone over the records of 476 mothers who had induction of labor with quinine in the Sloane Hospital. Sixty per cent of these mothers returned to the pediatric clinic with their babies. A questionnaire was sent to 374 of them, and replies were received from 65. None of the children who were actually examined or on whom the mothers reported had deafness which could

be attributed to the quinine. Eight had had otitis media. It is a fair assumption that the children of the mothers who did not reply to the specific questionnaire showed no abnormality of hearing. In my private practice there is no child with deafness.

My conclusion, therefore, is that while there is experimental evidence proving the passage of quinine across the placental septum to the child in utero—which may lead to a considerable concentration of the drug in the latter—there is not sufficient clinical evidence that this may cause either the death of the child or permanent injury to the auditory apparatus to warrant discontinuing the use of this valuable drug in obstetric practice.

Conclusive evidence could be obtained only by a comparison of the number of cases of so-called congenital deafness in a large series of children born after the administration of quinine to the mother with that in a similar series of children of mothers to whom no drug had been given. This evidence is still lacking.

NERVE DEAFNESS FROM SYPHILIS (CONGENITAL AND ACQUIRED). DR. ANTONIO CIOCCO, Washington, D. C.

A review of the extensive literature concerning syphilis of the eighth cranial nerve shows that the auditory defects most commonly recorded are (1) loss of acuity for high tones and a lowered upper tone limit, (2) loss of acuity for all tones by air conduction and impaired bone conduction and (3) shortened bone conduction associated with "normal" hearing by air conduction. Since with the exception of the last these defects constitute the classic signs of "nerve" deafness, it is important to determine whether or not these particular defects are found more frequently in syphilitic patients; and if there are any forms of "nerve" deafness, it is important to determine whether or not these particular defects are found more frequently in syphilitic patients, and if there are any forms of "nerve" deafness which may be regarded as pathognomonic of syphilis. The material studied included all hospitalized patients tested as part of the routine of the laboratory. My observations may be summarized as follows: 1. Syphilis apparently does not cause "nerve" deafness more often than any other general systemic disease, nor is there any particular type of "nerve" deafness found more frequently in syphilitic patients. 2. When impairment of hearing does occur it is found more frequently in persons with neurosyphilis. 3. Antisyphilitic treatment is apparently neither deleterious nor beneficial in the majority of cases.

DISCUSSION

C. C. BUNCH, PH.D., St. Louis: A number of years ago my associates and I reported a study of the hearing of a group of hospitalized patients, some of whom had syphilis but whose hearing was normal. In this earlier study the records of patients who were hard of hearing were eliminated when the averages were computed. This must not be interpreted as indicating a belief that syphilis cannot produce deafness. The paper just read indicates a similar contention—many patients with syphilis have normal hearing.

Syphilis does produce deafness at times. Here (slides) are the audiograms for 2 cases of deafness in which it is assumed that syphilis was the etiologic factor because of the improvement which took place under antisyphilitic treatment. In the first case the hearing gradually returned to normal in one ear, remaining unchanged in the other under antisyphilitic treatment. In the other case the patient was not entirely cooperative in the treatment, and the hearing improved little.

Dr. Ciocco's study is of intense interest from the statistical point of view and shows the careful manner in which he has approached the statistical analysis of the hearing of his patients.

DR. ANDREW A. EGGSTON, New York: For many years the Manhattan Eye, Ear and Throat Hospital has maintained a clinic devoted to the treatment of syphilis as observed in the eye, ear and throat. All the patients are followed from year to year by the department of social service, which constantly insists that

they return for regular systematic injections of arsphenamine and compounds of mercury or bismuth. As a result, some of the patients have been given as many as three hundred injections.

Two illustrative cases are presented. The first was that of a private patient of Dr. Fowler Sr., whom he had known personally for years. In 1926, when the patient was 46 years of age, there was a great change in his personality concomitant with loss of hearing. Thorough examination included Wassermann tests of the spinal fluid and the blood, a four plus reaction being obtained for both. His mentality was so affected that an eminent authority said he would be hopelessly insane in a year. The treatment was two intravenous injections of arsphenamine and, over a period of eleven years, sixteen of neoarsphenamine and seventy of arsphenamine. His mentality improved, and today he is conducting a large and successful business. His audiogram shows no improvement in the hearing of the lower or high notes, but certainly there has been a noticeable improvement of the bone and air conduction of the tones of the intermediate zone.

The second case was that of a clinic patient, aged 40, with nerve deafness. Treatment began in 1929; no concurring neurologic symptoms were noted, and the Wassermann reaction of the blood was positive. In eight years he has received injections as follows: twenty-eight of neoarsphenamine, sixty-one of compounds of mercury, sixty-six of silver arsphenamine and ninety-six of compounds containing bismuth. His audiogram shows no appreciable improvement in spite of all the injections and in spite of the fact that he thinks he can hear normally.

My associates and I have observed that patients with concurrent cerebral involvement seem to show better effects from treatment than those in whom the lesion is primarily in the nerves or the end-organ of hearing. The drugs of choice for treatment of syphilis of the eye and ear are silver arsphenamine and bismuth. We are convinced that silver arsphenamine is the safest drug; no convincing evidence is available that arsenic in this form has injured any patient, and certainly in cases of syphilis involving the optic nerve the silver radical appears to have the best effect and should be the drug of choice.

NERVE DEAFNESS FROM INFLAMMATORY LESIONS (OTITIS MEDIA, PETROSITIS, MASTOIDITIS, LABYRINTHITIS AND MENINGITIS, WITH OR WITHOUT NECROSIS). DR. SAMUEL J. KOPETZKY, New York.

This paper presents the process by which nerve deafness comes about, the types of bone in relation to the frequency of occurrence, the tissue reactions that take place around the windows in the labyrinth and the mechanics of labyrinthine invasion, with its resultant destruction of acoustic nerve tissue along pathways created by erosion. No matter what the causative factor, the picture within the labyrinth in all these conditions is the same. It varies in intensity, owing to the kind and nature of the irritant. It primarily takes the form of hydrops labyrinthi without fibrin content. The suppurative or necrotic lesion in the tympanomastoid or in the meninges first produces a serous or serofibrinous exudate in the perilymphatic space. This is a protective inflammatory secretion and is probably designed to ward off any purulent process seeking entrance to the internal ear. The endolymphatic space in turn is irritated by this secretion in close proximity to it, and hypersecretion of endolymph results. That it is not a direct reaction to the presence of bacteria seems established.

Hydrops labyrinthi shows itself in the endolymphatic spaces and occasionally in the perilymphatic spaces. This reaction within the labyrinth holds the key to the destruction of the nerve which is under consideration. It is a complication of acute tympanomastoid infection. Its occurrence is enhanced when the tympanomastoid areas are lined with strongly hyperplastic tissue. Hence, it is found to occur in the acute exacerbations of chronic disease, for chronic disease of the middle ear produces hyperplastic tissue.

If the antecedent causative factor is eradicated, the hydrops disappears as the irritant is removed, to reappear if the causative factor reappears.

Hydrops labyrinthi is the initial stage of all inflammatory reactions in the ear. Furthermore, from experimental data it is evident that when promptly relieved, it recedes, is absorbed and leaves little trace of its presence. The various purulent lesions obviously cannot heal with similar results. They leave scars and destruction of tissue in their wake.

The duration and the intensity of the hydrops labyrinthi determine whether or not the nerve elements of the membranous labyrinth shall survive. Hence, it is evident that the essential factors which produce hydrops labyrinthi should be better understood and whenever possible obviated. When the end-organ of the acoustic nerve in the membranous labyrinth is destroyed, whether by pressure exerted by the nonpurulent fluid of hydrops or by frankly purulent matter, there is no therapy, surgical or nonsurgical, that I know which will restore it to useful function.

DISCUSSION

DR. JOHN R. PAGE, New York: There is little of value or interest to add to Dr. Kopetzky's paper except perhaps from a clinical standpoint through reference to a case that came under my observation some years ago (*Ann. Otol. Rhin. & Laryng.* **27**:589 [June] 1918) that illustrates reasonably how it is possible for infection to extend from the middle ear and the mastoid along the facial canal and produce retrolabyrinthine nerve deafness on both sides, with total loss of hearing in the opposite ear and partial loss in the ear from which infection started. There was first facial paralysis, then meningitis, then total deafness of the opposite side. Three days after a simple mastoidectomy was performed for acute involvement of the right mastoid in a boy 10 years old, beginning facial paralysis developed on that side which was complete in forty-eight hours. Seven days after the operation total loss of hearing in the opposite ear occurred suddenly, with vomiting and a roaring tinnitus but without nystagmus or vertigo. A high leukocyte count (42,000) and cloudy spinal fluid in which there were no bacteria were present; the mental condition remained rational almost until his death, on the tenth day after the operation. Until the sudden total loss of hearing in the opposite ear it was not realized that there was also marked impairment of hearing in the ear that had been recently operated on. It is conceivable that the auditory nerve of this ear became involved at about the same time as the facial nerve on that side through its close association with that nerve in the internal auditory canal and that involvement was not so extensive that it destroyed the hearing entirely. It would seem that if invasion of the meninges had taken place through the labyrinth on this side there would have been vertigo and nystagmus and total loss of hearing. The sudden total loss of hearing in the opposite ear with roaring tinnitus and vomiting leads one to think first of the labyrinth; when it is recalled, however, that from first to last the patient was able to sit up without disturbance of equilibrium, it seems improbable that any destruction of the labyrinth could have taken place, for while simultaneous obliteration of both labyrinths might occur without nystagmus, it could not well occur without total deafness, which was not present in this instance.

In this case it seems that the nerve deafness and persistent vomiting were caused by meningitis and not by disturbance of the labyrinth and that invasion of the meninges took place along the route of the facial nerve, causing paralysis and also marked impairment of the auditory nerve in its course.

Of some clinical interest also is the proportion of cases in which symptoms of acute labyrinthitis develop after suppuration of the middle ear, both acute and chronic, in which there is temporary or permanent total loss of both static and auditory functions or a permanent total loss of the auditory function with the static labyrinth active. I do not recall having observed a case of labyrinthitis associated with suppuration of the middle ear in which the reverse happened and there was an active cochlea with a dead static labyrinth. Several years ago when I looked up the subject, it was not possible to find in the literature the percentage of cases in which diffuse labyrinthitis that developed from suppuration of the middle ear and the percentage in which the process either

resolved, with or without some return of function, or spread to the meninges. Braun and Friesner were quoted at that time, with Friedrich and Hinsberg as their authority, as saying that there is 1 case of labyrinthitis for every hundred cases of suppuration of the middle ear. The hospital records examined at that time were so far short of that number that with the help of Dr. Fowler's clinic and my own 229 cases of suppuration of the middle ear of all kinds were collected in a few weeks. Ninety-three patients had had acute abscess of the middle ear, and 20 had undergone simple mastoidectomy, 7 modified radical mastoidectomy and 28 radical mastoidectomy. Among the 81 persons with chronically suppurating ears, 1 had a totally dead labyrinth and 3 had active static function but a dead cochlea. The labyrinths were active in all the others except 3 who had undergone radical mastoidectomy and had lost the auditory but not the static function.

Altogether, in 229 cases there were 1 totally dead labyrinth and 6 dead cochleas. There were 3 dead cochleas with active static function in 81 patients with chronic suppurative disease of the middle ear who did not undergo radical mastoidectomy, and 3 dead cochleas with active static labyrinths in 28 persons who had undergone radical mastoidectomy.

In the light of some of Dr. Fowler's experiments with different solutions in the middle ear, it is surprising that there is not more damage to the cochlea from traumatism around the oval and round windows and from the use of chemicals over the inner tympanic wall, even when the greatest care is exercised in the radical mastoidectomy and the after-treatment.

NERVE DEAFNESS FROM NONINFLAMMATORY LESIONS IN THE TEMPORAL BONE. DR. EDMUND P. FOWLER JR., New York.

Osteitis Deformans.—This disease produces nerve deafness by stretching the nerve and constricting the vessels leading to the labyrinth and also by direct invasion of the internal meatus, the cochlea and the modiolus. Its progress can occasionally be controlled by roentgen therapy. This suggested that radiotherapy be used, especially in the early stages of the disease. Tests for phosphatase are a help in diagnosis.

Otosclerosis.—Nerve deafness occurred in some form in 38 per cent of the 126 cases studied clinically, usually in older persons. Occasionally, a growth invades the lower turns of the cochlea, and occasionally it grows into the internal meatus or the round window. This may lower the bone conduction. I cannot substantiate the finding of Gray that otosclerosis is due primarily to disease of the nerve.

Tumors.—Cholesteatoma, carcinoma and sarcoma all invade the labyrinth, as does neurofibroma. The common neurofibroma in the eighth cranial nerve grows on a pedicle from the base of the internal auditory meatus and into the classic cerebellopontile angle.

Fracture.—Fracture causes hemorrhage into the cochlea and so causes deafness and loss of cochlear function.

DISCUSSION

DR. HARRIS P. MOSHER, Boston: Dr. Fowler Jr's. paper covers the assigned subjects briefly and adequately. The author can discuss few subjects from actual clinical experience and must rely on textbook knowledge. Those present will undoubtedly agree with the recommendation that the percentage of phosphates should be watched in Paget's disease and that roentgen therapy is worth using as a preventive when this disease is suspected.

It would be unfortunate if it should be proved that loss of teeth will cause fracture of the cribriform plate, as that would make a great many persons nervous as they leave this meeting.

This question of the loss of teeth, pain in the ear and deafness has been brought up as new. It is not new. It has been in the literature for the last ten years.

Of particular interest is the clearcut contrast which Dr. Fowler Jr. brought out between Paget's disease and otosclerosis. Many, however, will feel a bit disappointed that the observations on 23 persons with otosclerosis in regard to the histologic changes in the eighth cranial nerve did not bear out the findings reported by Gray. Gray was such a splendid observer as well as such a splendid man that it comes a little hard to disagree with him. The explanation that Gray's findings were due to faulty autopsy technic treats the matter a little too lightly.

I can agree from personal experience with Dr. Fowler Jr's. remarks about cholesteatoma and new growths involving the eighth cranial nerve.

In regard to osteoma inside the skull, I can say a word from personal experience also. For some years I have been watching skulls as they came from the dissecting room, and osteoma was found to be fairly common. It occurs most commonly on the posterior surface of the frontal bone, next in the middle fossa and third around the internal auditory meatus. I have never seen one in the posterior fossa.

NERVE DEAFNESS FROM CENTRAL AND CORTICAL LESIONS: PARTIAL SECTION OF THE EIGHTH CRANIAL NERVE. DR. WALTER E. DANDY, Baltimore.

From a series of intracranial operations on human subjects, which serve as experiments, I wish to call attention to certain facts concerning the effects on hearing of operation in two different lines in the pathways of hearing.

(Slide) This sketch shows what one will see in any textbook, with the exception of the central pathways for hearing, i. e., those in the cerebral hemispheres.

It is universally claimed that the center for hearing lies in the temporal lobe. How this conception has arisen is difficult to understand, but it is certainly a mistake, for one can remove either the entire right or the entire left temporal lobe with no effect whatever on hearing. Audiometric tests have, of course, been used in checking these experiments.

Another more extensive experiment is perhaps even more impressive. When the entire right cerebral hemisphere is removed (this has been done seven or eight times), there is not the slightest effect on hearing.

After removal of the occipital, frontal or temporal lobe on the left side (in different persons), there is no effect on hearing. Therefore, the only possible location for a center of hearing is in the left parietal lobe. It is unquestionably connected with the auditory speech center, which is located in the parietal lobe and not in the temporal lobe as most textbooks state.

In this connection two interesting points from a negative standpoint are (1) that between the mesial geniculate body and the inferior colliculus anteriorly and the point of entrance of the eighth cranial nerve into the brain stem posteriorly I have never seen a tumor that has produced deafness, and (2) that I have never seen a tumor in the cerebral hemisphere, right or left, that has produced deafness unless there has been an associated deficiency of the auditory speech centers. These facts must carry significance, although I am not prepared to make a positive explanation. Possibly there may be one central region in which defects of hearing may carry diagnostic significance, i. e., at the inferior colliculus or the medial geniculate body. I have observed 3 cases in which tumor in this region (pineal or adjacent) has produced an abrupt drop in the hearing for high tones by both ears. These may have been purely coincidental findings, but my belief is that they are significant. In 1 case in which an audiometric test had not been made and the tumor was found in this location, a subsequent check-up disclosed this same suspected finding.

Other than this, when deafness results from an intracranial tumor, the lesion is always located along the eighth cranial nerve itself. It is therefore of the greatest and most certain diagnostic import.

Regarding the absence of defects of hearing from tumor throughout the brain stem, my only explanation is that the fibers scatter after their arrival in the brain stem.

My next point of experimental attack is in the eighth cranial nerve itself, i. e., in the intracranial portion between the spiral ganglion and the brain stem.

In a large series of operations for Ménière's disease my associates and I have made one interesting observation—one can divide from one half to two thirds, at least, of the auditory division of the eighth cranial nerve and lose practically no hearing. When a slight loss does occur, it is restricted to the extremely high tones.

In Ménière's disease, in which deafness is always associated with the attacks, the lesion must be in the nerve itself. In several specimens removed at operation there has been fibrosis of the nerve. This is the explanation of the syndrome. It is the only intrinsic change I have been able to find. In about 20 per cent of the cases a large artery (one of the branches of the superior-inferior cerebellar artery) lies against the nerve and is probably one of the causes of the deafness and dizziness which are a part of this disease.

DISCUSSION

DR. R. LORENTE DE NÓ, New York: The phenomenon reported by Dr. Fowler Sr. may be described in the terminology used by physiologists in the following manner:

As a consequence of unilateral lesions (or of lesions more pronounced on one side) in the neural mechanism of hearing, there appears a difference in threshold for the two ears. The difference is diminished and even disappears when the strength of the stimulus is increased. Hence, the difference of threshold between the normal (or less affected) ear and the diseased (or more affected ear) is not constant, but depends on the strength of the stimulus. Dr. Fowler Sr. has also determined that the phenomenon does not occur in conduction deafness.

A consideration of facts known about the anatomy and physiology of the nervous system has revealed a simple explanation.

Every fiber in the cochlea is connected with several cells, and every cell is connected with several fibers. On the other hand, the only type of message which the nerve fibers carry to the centers is made up of impulses, comparable to the pulses of current released by the key of a telegrapher. The nerve fibers cannot carry impulses at a frequency higher than a determined limit. Furthermore, it is known that one, and perhaps the most important, of the variables determining the intensity of a stimulus is the frequency of the impulses carried by the nerves. In general, it may be said that the greater the frequency, the greater the intensity of the stimulus.

From this it follows that as there is a limiting frequency of impulses, analogously, there is also a limiting intensity for each localized stimulus.

If a number of hair cells in the ear or a number of fibers in the cochlear nerve are missing, the tones will appear to be weaker when stimuli near threshold are used, but if the intensity of the tone is increased, the more strongly activated hair cells or cochlear fibers will be sufficient to saturate, i. e., to excite with the limiting intensity the cochlear fiber or the cells of the cochlear nuclei, so that the cerebral cortex will receive the same number of impulses per second from both ears and will perceive the tone delivered to the diseased ear as strongly as the tone delivered to the normal or less affected ear.

Thus, it may be said that Fowler's phenomenon is an immediate consequence of the anatomy and physiology of the nervous system and that in fact it must be pathognomonic of neural deafness.

NERVE DEAFNESS FROM CENTRAL AND CORTICAL LESIONS. DR. HAROLD G. TOBEY, Boston.

Tumor of the cerebellopontile angle is a frequent cause of unilateral partial deafness.

Anomalies of the vessels are proved to be somewhat frequent causes of Ménière's disease. It has been pointed out that the symptoms of Ménière's disease are the same as the early symptoms of tumor of the cerebellopontile angle.

The part played by tumor of the brain as a cause of deafness is still somewhat uncertain. The anatomy of the auditory tract has been well worked out, and

the study is nearly complete. The auditory center is anatomically localized in the superior temporal convolution, particularly in the transverse gyri of Heschl.

The physiologic experimental evidence as to the exact location of the auditory center is beset with contradictions.

From the more recent literature the consensus seems to be that deafness is not a common symptom from tumor of the brain.

DISCUSSION

Dr. David Rioch, Boston: The difficulty of diagnosis of lesions of the central acoustic centers and pathways is obviously due for the most part to the extensive crossing in the system which leads to an almost equal bilateral representation of each cochlea at all levels above the acoustic nuclei and the trapezoid body. Further difficulties arise from ignorance of the functions of the various parts of the system. It may therefore be of interest to recall certain observations made on animals with extensive lesions of the brain. In chronically decerebrate cats Bazett and Penfield noted slight movement of the head in response to a high-pitched sound of low intensity. Bard described two types of response to auditory stimuli in cats which survived removal of the neocortex and archicortex and most of the striatum and the thalamus. In response to a soft high-pitched sound, the head was elevated, the ears were pricked up, the palpebral fissures widened and the animal turned in the direction of the sound. Localization was accurate, and one cat even followed the sound around the room. To a loud, high sound (e. g., a blast on a bugle) the animals reacted in a manner indistinguishable from the expression of fear in a normal cat. One animal with a more extensive lesion involving both medial geniculate bodies failed to respond to any auditory stimulus further than by twitching the skin of the back. Bard's observations have been confirmed in the laboratory at the Harvard University Medical School, with minor variations in cats surviving removal of the neocortex. In addition, it has been shown that a response exactly simulating that to a soft, high-pitched sound can be evoked by electrical stimulation of the lateral surface of the remaining part of the brain stem.

Such observations are consistent with anatomic data, which have demonstrated extensive connections of the acoustic system with subcortical centers.

Hearing Aids for "Nerve Deafness." John C. Steinberg, Ph.D., New York.

Results reported by Dr. Fowler Sr. at last year's meeting on tests of hearing with sounds of above threshold intensity showed two types of deafness. In one type the deafened ear hears less loudly than the normal ear at all levels of intensity, but in the other type the deafened ear hears loud sounds with practically the same loudness as the normal ear. Similar results have been observed by my associates and me in our studies at the Bell Telephone Laboratories. The occurrence of these two types of deafness can be explained on the ground that the loudness of a sound is closely related to the total number of nerve fibers conducting impulses from the ear to the brain, owing to the stimulation by the sound. This point of view indicates that the type of deafness in which loud sounds are heard normally may be caused by nerve lesions and the type in which all sounds are heard at reduced loudness may be caused by a lesion in the middle ear. It is shown that the amplification of sound for the ear with conduction deafness tends to restore faint and loud sounds to their normal loudness relations. For the nerve-deafened ear, however, amplification overemphasizes the loud sounds in relation to the faint sounds, and if great enough may make them uncomfortably loud.

DISCUSSION

Dr. Horace Newhart, Minneapolis: It is hoped that the facts discovered by Dr. Steinberg and his associates and Dr. Fowler Sr. concerning the relation between increased loudness and amplification of the higher frequencies, as perceived by persons having nerve deafness may be a step toward providing more effective electrical hearing aids. Dr. Steinberg admits that this cannot be realized

immediately because of mechanical and other engineering difficulties to be overcome. Selection of hearing devices is a difficult problem for otologists, especially when there is a great loss of hearing for the higher frequencies. The ordinary carbon microphone is unsatisfactory because of distortion.

Selective amplification has materially increased the application of portable hearing aids. Nevertheless, severely deafened persons may need instruments with vacuum tube circuits, using maximum amplification with minimum distortion. Such instruments are nonportable or semiportable. Marked improvements have been made recently in the efficiency of the semiportable instruments by reduction in size and increased fidelity. They should be more frequently prescribed by the otologist.

My attention has been forcibly called to a new instrument of this type by the enthusiasm of several patients. The performance of this instrument is familiar to me from actual investigation. Others of similar type will doubtless be produced by other manufacturers if they are not already on the market. The features of this particular hearing aid include a high grade microphone, a crystal receiver, tone and volume controls giving a wide variation of both tone and volume, an induction telephone attachment, which amplifies telephone conversation, and an attachment whereby the user can operate the instrument at will by the regular electric light current, thus obviating the carrying of heavy batteries. This will prove a great boon to patients with severe nerve deafness, who cannot be helped by the portable hearing aids now available.

(To be concluded)

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. H. Burger, Keizersgracht 317 C, Amsterdam.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Atlantic City, N. J.

American Laryngological Association

President: Dr. J. F. Barnhill, Miami Beach, Fla.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Atlantic City, N. J.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Samuel J. Kopetzky, 71 E. 80th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: April 27-29, 1938.

SECTIONS:

Eastern.—Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Philadelphia. Time: Jan. 7, 1938.
Southern.—Chairman: Dr. Murdock S. Equen, Medical Arts Bldg., Atlanta, Ga.
Place: Atlanta, Ga. Time: Jan. 24, 1938.
Middle.—Chairman: Dr. James B. Costen, Beaumont Bldg., St. Louis.
Place: St. Louis, Mo. Time: Jan. 26, 1938.
Western.—Chairman: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Place: Santa Barbara, Calif. Time: Jan. 30-31, 1938.

American Otological Society

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Atlantic City, N. J.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.